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VOL. XLI

MARCH, 1932

No. 1

Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING

THE INDEX OF OTOLARYNGOLOGY

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PUBLISHED QUARTERLY

BY THE ANNALS PUBLISHING CO.

7200 WYDOWN BLVD.

ST. LOUIS, MO., U. S. A.

SUBSCRIPTION PRICE, \$6.00 PER ANNUM, IN ADVANCE

Subscription price in Canada, \$6.40.

Subscriptions in other countries of the Postal Union, \$6.80.

Entered at the Postoffice, St. Louis, Mo., as Second-class Matter.

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MARCH, 1932.

No. 1.

I.

MALIGNANT DISEASES OF THE NOSE AND NASAL
ACCESSORY SINUSES.*

JOSEPH C. BECK, M. D., AND M. REESE GUTTMAN, M. D.,

CHICAGO.

This presentation will deal with the neoplasms of the nose and nasal accessory sinuses. It must be borne in mind that it will be somewhat difficult to sharply limit the discussion to this region, as undoubtedly neoplasms having their origin in the nose or nasal accessory sinuses may, by their progression invade the orbit or nasopharynx.

It should also be brought out at this time that the discussion will be brief and that details will be, to a more or less degree, omitted, as the time allotted to the discussion is very short. It might be well to open this subject by presenting and discussing a series of histopathologic specimens of various neoplasms that are commonly observed about the nose and nasal accessory sinuses.†

*From the Symposium on Malignant Diseases, presented before the American Academy of Ophthalmology and Otolaryngology, French Lick, Indiana, September 14, 1931.

†Forty illustrations are shown on the screen, and an example of one is shown in Fig. 1.

The first type of neoplasms to be discussed are various so-called precancerous entities. These are, in the main, nonmalignant pathologic conditions, which very frequently form a substratum upon which a malignancy is grafted. One of the very common ones is rhinophyma, a pathologic state that is not in itself malignant but very frequently undergoes malignant degeneration, due to the ingrowth and proliferation of epithelium in the pathologic overgrowth of the glandular tissue of the skin of the nose.

Paraffinoma.—Paraffinoma of the skin about the nose and face, due to the injection of paraffin for cosmetic purposes, is another benign condition that may at times assume malignant characteristics. Most frequently the type of malignancy that follows is of a sarcomatous nature, due to the fact that the irritation is in the connective tissue rather than the epithelium.

Various other benign conditions were exhibited upon the screen showing their histological characteristics, upon which a malignancy might be engrafted.

Cancroid.—One of the most important common dermal lesions of a malignant nature is the cancroid, (Fig. 1.) Microscopic study will show that the basement membrane has been broken through and invaded by epithelial cells. The malignant nature of these cells is well shown by the fact that they vary in size and shape and also in their staining characteristics. One may also note the presence of numerous mitotic figures. Careful examination of the section will show that in the periphery of the lesions there is a great deal of fibrosis, and that this fibrosis, in a manner of speaking, chokes off the lymphatics of the skin and accounts for the very slow spread of cancroid lesions.

The histologic appearance that is exhibited by a cancroid is of practical importance, as it is a matter of common knowledge that this type of lesion responds very favorably to radium treatment. In other words, this is what is known as a radiosensitive growth. It is well known that as many as 90 per cent of the malignancies of this type can be permanently cured by early irradiation alone.

At this time a discussion of the value of the microscopic study of the neoplasm in relation to the diagnosis, course, prognosis and treatment of the disease, is in order. It is only since the recent

collaboration of both the pathologist and the radio histologist that one has been able to gain some information regarding the value of the minute picture of the neoplasm as regards the course that the disease pursues and its response to therapy. It has been found that cellular types of neoplasms, which clinically are so highly malignant, and metastasis so readily and extensively are very frequently the most radiosensitive. For instance, it has been found that neoplasms that are embryonal in character, so-called anaplastic lesions, that are highly cellular, and show no tendency towards adult structure, are the ones that are most easily influenced by radio therapy, whether radium or X-ray. And yet, it is also peculiar that the lesions that show a tendency towards adult characteristics—for instance, the acanthotic lesions of a squamous cell carcinoma, that show pearl formations and regular pavement-like tissue structure—respond very poorly to irradiation. These are best handled by extensive surgical destruction.

Cellular sarcomas, such as small round cell sarcomata and lymphosarcomata, respond very readily to irradiation. On the other hand, fibrosarcomata, carcinomata having a desmoplastic reaction or fibrous infiltration, respond poorly, even to large doses of radium or X-ray. It should also be stated that while the Broder classification of the degree of malignancy is a worth-while study, yet in clinical practice we have been unable to find any correlation between the classification as given by him and the actual clinical manifestations of the disease.

Biopsy.—The question of biopsy is a very important one. Biopsy in the nose, throat and pharynx is comparatively easy, as the neoplasm, as a rule, is accessible. One should remove a sufficient amount of tissue in order to enable the pathologist to make a good preparation so that it can be easily studied. The question of metastasis after biopsy is, of course, a vital one, but we feel, as does Dr. Jaffee of the County Hospital in Chicago, who said, "A biopsy performed with a minimum of trauma, using a cold sharp blade, has very little influence on metastasis." However, in order to safeguard any such occurrence, we have been insistent in telling our patients that should the return of the microscopic section be reported as a malignancy, then we advocate immediate surgical or radiological intervention.

We have come more and more to the point of view held by Dr. New, namely, performing a biopsy on the operating table, getting the report of the frozen section and then proceeding immediately with radiation, or surgery if it is found necessary. The biopsy, however, must be carefully guarded from the moment it is removed from the operating room until it has undergone the technical treatment by the technician. On rare occasions biopsy material has unfortunately been exchanged and a wrong diagnosis returned to the surgeon. Such carelessness is to be deprecated, but unfortunately, as has been stated by Dr. Bloodgood, it has occurred in a few instances.

The gross diagnosis of a malignancy of the nose and nasal accessory sinuses, as a rule, is not at all difficult. The lesion is there, available for inspection, and even palpable with a probe. However, one must always bear in mind that the clinical differentiation between the various granulomata may at times be not only difficult but also impossible.

We have assumed the dictum that whenever a malignancy is diagnosed or even suspected, a biopsy is always imperative as a confirmatory measure. One may have other signs and symptoms to aid in the establishment of the diagnosis. The X-ray showing peculiar types of encroachment upon the bones of the face, aided by the use of radiopaque oils; may frequently be of help. Such confirmatory evidence as enlarged glands in the neck and the presence of cachexia may also be of assistance. It should be once more reiterated that the diagnosis most frequently rests upon the biopsy and study of the histologic section. The gross pathology of these various conditions is, of course, best studied either on the patient at the time of examination, in the operating room and at times only possible at postmortem.

There is herewith appended a table of malignant disease about the nose and nasal accessory sinuses. (Fig. 2) as observed by us. This is only a partial list of patients actually seen and refer only to those of which we have a complete clinical record and, as a rule, some photographic evidence—for instance, photographs of patients showing evidence of gross involvement, the progress, as well as the gross specimens, were shown on the screen. An ex-

ample of such illustration is shown in Fig. 3, up to and including Fig. 26.

Many other cases seen in clinical practice at the County Hospital and in consultation, of which no records are available, are, of course, not listed.

Prognosis.—The prognosis of malignant disease about the nose and nasal accessory sinuses is not a very happy one. Even with our modern means of attack, namely, the use of the electrothermic methods of surgery, aided and abetted by the use of radium and X-ray, our percentage of cure leaves a great deal to be wished for. The fact that the anatomic situation unfortunately is in close proximity to vital structures and also that bone is so frequently involved, accounts for this very sorry spectacle that the medical profession has to offer the laity. In contrast to this gloomy picture one should consider that about 90 per cent of the skin epitheliomas, whether basal or squamous cell in nature, respond, as a rule, providing the lesion is seen early, to irradiation.

Treatment.—The treatment of malignancies about the regions under discussion will, of course, vary with the histopathologic nature of the disease. Those highly cellular anaplastic, embryonic types of tumors that tend to metastasis so quickly, as a rule respond poorly to our surgical attempts, and it is in these cases that we may expect something in the future from the use of radium and X-ray. The relatively benign lesions, as shown by histopathology, may at some time respond to surgical intervention.

Surgery.—There have been several types of surgical approach to malignancies about the nose and nasal accessory sinuses advocated. One of the most common and most popular is the Moure operation (Fig. 27) or the Denker operation (Fig. 28). In the latter the exploration and resection is performed under the lip. In lesions that involve the ethmoid and frontal, the modified Lynch operation that permits a radical procedure without too much deformity is advocated, in contradistinction to the Preysing operation (Fig. 29), which, however, gives a wider exposure. The oldest, and, of course, most common procedure that has been utilized to attack malignancy of the upper jaw has been the

Kocher (Fig. 30) resection of the superior maxilla. With all of these operative measures, it is well to utilize X-ray or radium both as a preoperative and postoperative adjunct.

Surgical Diathermia.—The discussion of surgical treatment of malignancy about the nose and nasal accessory sinuses would, of course, be incomplete without some reference to electrothermic methods. We have been well satisfied by the use of the diathermic cutting current in performing our dissections. In addition to that, we use the coagulating current. In the first place, we employ it to throw a cofferdam of coagulation about the periphery of the growth if it can possibly be performed. We insert the coagulating needle into the uninvaded tissue at some distance from the edge of the advancing lesion and coagulate a regular circumscribing fence about it. Following this the tumor itself may be scooped out by using the cutting loop, or the tumor itself may be coagulated by using the various button or needle electrodes (Fig. 31) and then resort to the use of a curette to remove the débris. By utilizing these few technical procedures the entire visible portion of the growth may be removed. At the termination of the operation or some time after the reaction has subsided we frequently employ a large amount of radium. In the use of electrothermic methods we would like to refer to a new combined apparatus that enables one to employ both the cutting and the coagulating current at one time. There are several other features of this apparatus that are noteworthy in the advance of the technique, and in this I refer to the sterilizable electrodes, the connecting cords, and also to the sterilizable knob that permits the regulation of the current by the operator himself to suit his immediate needs.

Radium.—The use of radium is a very important advance in the treatment of malignancy. A great deal of progress has been made in this, not only in the technic of the use of the radium but also in the study of the relation between the histological structure of a tumor and its response to irradiation. You are all familiar with surface contact and interstitial use of radium (Figs. 32-33), but I would like to take this opportunity of calling your attention to a new advance in the technic of radium therapy, namely, teleo-radium therapy or teleo-Currie

therapy (Fig. 35), as it is called in Europe. In this new technical advance, one utilizes the so-called radium bomb or cannon. (Fig. 35 B.) A large amount of radium, up to four grams, is introduced into a heavy lead container, and the rays from this tremendous amount of radium are permitted to act upon a tumor at some distance from the skin. One may utilize the ray in exactly the same manner as the X-ray therapist, namely, by employing several portals of entry about the skin, all the rays of which are focused upon the tumor lying beneath (Fig. 35 C). Thus one can see that the tumor itself receives a tremendous dose of radium (Fig. 35 E), and the overlying skin only has small individual doses given at the various portals of entry. (Fig. 35 D.) Various technical methods are employed by which the radium is centered exactly upon the growth (Fig. 35 A), and the best results obtained. If one will spend any time perusing the literature emanating from the Radium-Hemmet in Stockholm and from the Regaud Clinic in Paris, as well as the various radium centers in this country, he will be surprised at the rapid progress being made and the results obtained by the use of radium in the treatment of malignant disease. In cases of cervical glandular metastasis, a favorite treatment is the radium collar, as illustrated in Fig. 34.

X-Ray.—There have also been several technical advances made in the use of X-ray therapy in the treatment of malignant disease. I have specific reference to the use of the new 900,000 volt X-ray tube (Fig. 14), just perfected by Dr. Coolidge, which has been installed at the Memorial Institute in New York. This apparatus at the present time is in the experimental stage and will be utilized in the treatment of malignant disease at that institution. However, physically speaking, the action of these X-rays under the high voltage tension of 900,000 volts, cannot approach that of gamma irradiation from radium. In order to produce a ray energy that is comparable to the gamma ray emitted by radium one may have to utilize three and one-half million to four and one-half million volts, and as yet these technical difficulties are apparently insurmountable, but probably not for long. The cost to install such a highly complicated apparatus and the large personnel will not, I fear, permit this method of treatment of malign-

nant disease to become very popular. In all probability the tendency will be toward the employment of more and larger doses of radium, although one must be cautious in predicting what the future will hold in store.

The type of deep X-ray therapy that we have employed on the cases we have shown you here today has been of an entirely different form from that mentioned of the extremely powerful machine that is still in the experimental stage. At the beginning of our experience the voltage was not any higher than about 180,000 volts. At the present time the apparatus that we are showing you is of a much higher voltage, 300,000 volts, and with it some very good results are recorded. However, thus far we have had no great benefit in using it on malignancies deep below the skin.

In conclusion, it must be emphasized that no one certain definite type of surgical operation, such as the Caldwell-Luc, or the Moure, or the Killian, is ever employed in the classical manner, but must be modified to suit the individual case as it occurs. It should also be stressed that in this region particularly, and with the use of electrothermic methods of destruction of the neoplasm, the question of hemostasis is of greatest importance. For that reason, in an attack on a neoplasm of any considerable size, we have always deemed it a precautionary measure to prophylactically ligate the carotid artery, most frequently the external, but under the stress of obscured anatomy within the neck due to secondary involvement, the common carotid has had to be ligated instead.

There are a number of other factors of importance that must also be stated. This is particularly true in inoperable cases with extensive metastasis in the neck and into distant parenchymatous organs. In these hopeless cases palliative measures are of prime importance. Pain may not at times be amenable to opiates, and one must, therefore, consider alcohol injections into the trigeminal nerve or even into the Gasserian ganglion, and at times it may be necessary as a measure of last resort to resect the posterior root.

185 North Wabash Ave.

(This is the first of a series of articles dealing with neoplasms about the nose, throat and ear, by Drs. Beck and Guttman. The second article will appear in the June issue.)

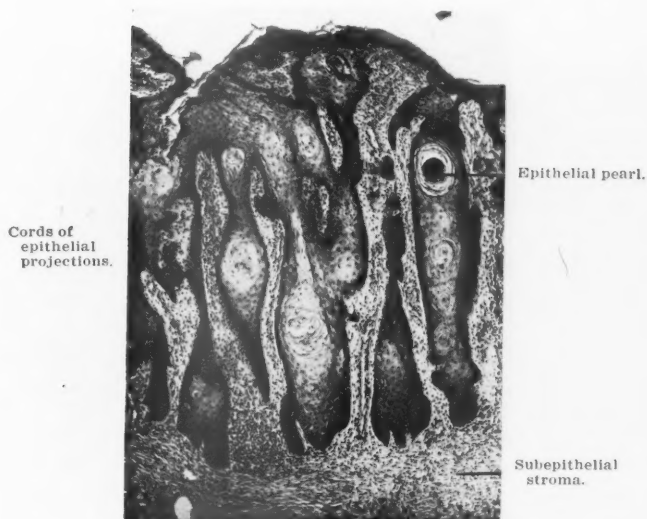


Fig. 1. Canceroid of external nose.

PARTIAL LIST OF NEOPLASMS OF THE NOSE AND NASAL ACCESSORY SINUSES.

	Cases
Rhinophyma	10
Rhinophyma verrucosa.....	2
Nevus angiomatosus	8
Adenoma	2
Cystadenoma	1
Paraffinoma	5
Hemangioma	14
Lymphangioma	1
Fibromyxoma (not nasal polypi).....	1
Fibrochondroma	2
Osteoma	13
Papilloma	19
Epithelioma	180
Cylindroma	2
Sarcoma	212
Chloroma	1
Plasmacytoma	1
Cholesteatoma	1
Rhinoscleroma	5
Tuberculoma	11
Syphiloma	45
Mucocystoma	8
Teratoma	2
Adamantinoma.....	1
Dentigerous cyst.....	21
Meningo and encephalocele	5
Dermoid cyst.....	2
Endothelioma	5
Total.....	579

Fig. 2.

MIXED OR NON-DIFFERENTIATED CELL SARCOMA, HIGH-
LY PIGMENTED, OF NOSE AND NASAL ACCESSORY
SINUSES COMPLICATED BY EXTENSION IN ALL
DIRECTIONS, INCLUDING GENERALIZED
METASTASIS.



Fig. 3. The growth begins to ulcerate near the center of the hard palate. The corresponding swelling over the left side of the face. Radium needles 200 milligrams inserted peripherally of growth, remaining for ten hours. Deep X-ray over left side of face.



Fig. 4. Two weeks later the growth has disappeared with formation of fistula and scars. The swelling on face reduced.



Fig. 5. Approximately one week later. Beginning recurrence with filling in of the fistula.



Fig. 6. Greater progress of growth.



Fig. 7. One week later, still greater progress, blocking the entire left side of the nose. Repeating radium needles and deep X-ray therapy, same dose as Fig. 3.



Fig. 8. Growth receding with greater destruction of hard palate.



Fig. 9. Two weeks later; swelling subsiding.



Fig. 10. Front view, showing still some swelling.

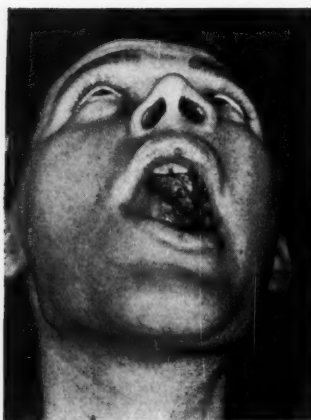


Fig. 11. Approximately one week later, growth showing rapid recurrence.



Fig. 12. Masses removed with partial resection of upper jaw.



Fig. 13. Showing cavity following resection.

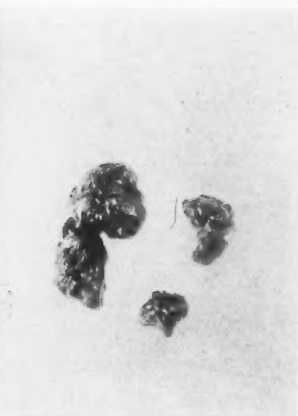


Fig. 14. Masses removed from cavity by radio knife and the remaining cavity thoroughly treated by electrocoagulation.



Fig. 15. Immediate placement of obturator to aid in feeding, speech and prevention of reinfection.



Fig. 16. Approximately one week later, rapid recurrence, showing growth protruding from cavity.



Fig. 17. Same, front view, showing extensive growth on the face. Complete nose block on left side.



Fig. 18. Masses removed at reoperation by curettage and subsequent electrocoagulation.



Fig. 19. Apparent standstill—no further growth. Patient losing rapidly in weight.



Fig. 20. Approximately two months later, recession of side of face. No evidence of growth.



Fig. 21. Acute osteomyelitis and cellulitis of the left side of mouth and face.



Fig. 22. Approximately two months later. Patient gaining in weight, though greater contraction over the left side of face.



Fig. 23. Approximately four months later. Patient in excellent general condition. No evidence of any recurrence.



Fig. 24. Beginning breaking down of skin covering the side of the face near nose. Small fistula at side of nose.



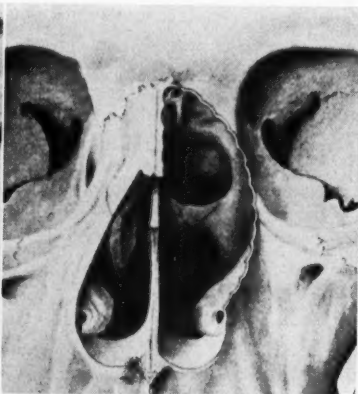
Fig. 25. Approximately two weeks later. Greater loss of substance over the side of face. Marked loss of appetite and weight. Rise in temperature.



Fig. 26. Approximately one week later. Rapid breaking down of the side of the face. General decrepitude.



(A) Incision.



(B) Complete evulsion.

Fig. 27. Moure's operation.

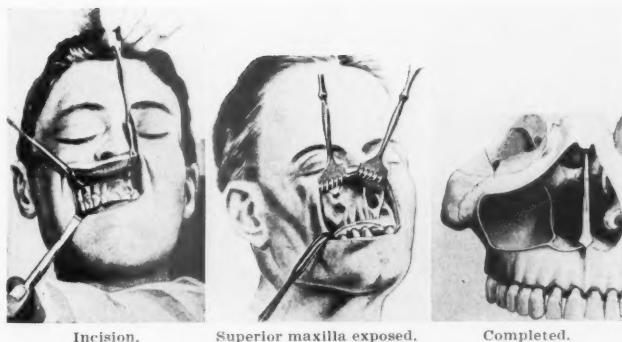


Fig. 28. Denker's operation.

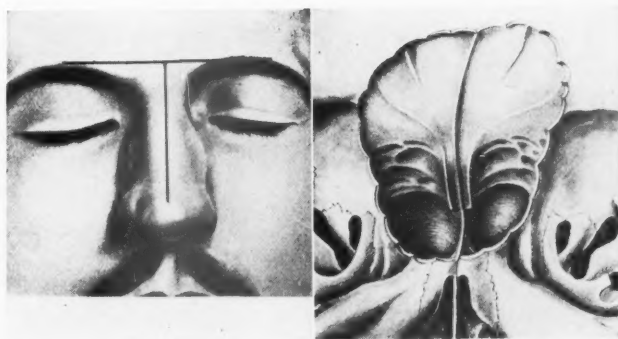


Fig. 29. Preysing Operation.

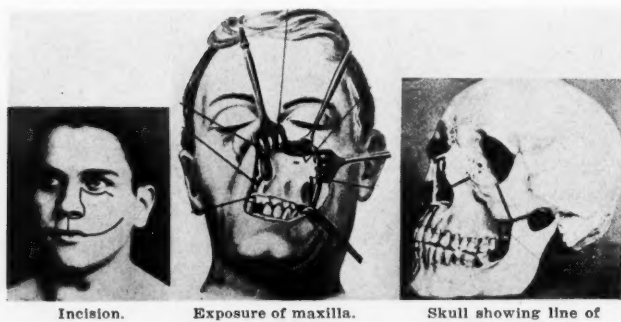


Fig. 30. Kocher's operation.

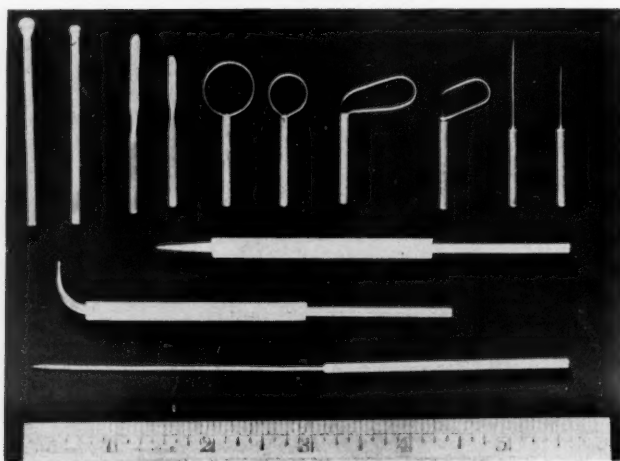


Fig. 31. Various endothermic electrodes.

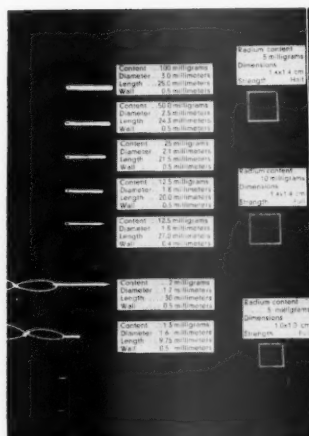


Fig. 32. Radium capsules, needles, plaques and radon seeds.

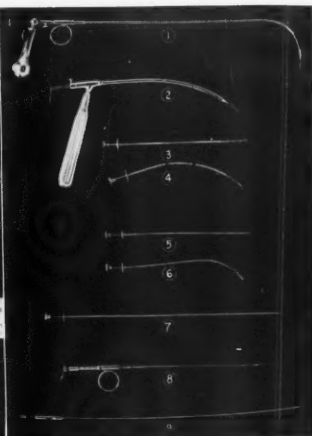


Fig. 33. Radium introducers used for neoplasms about the head and neck.



Fig. 34. Radium collar.

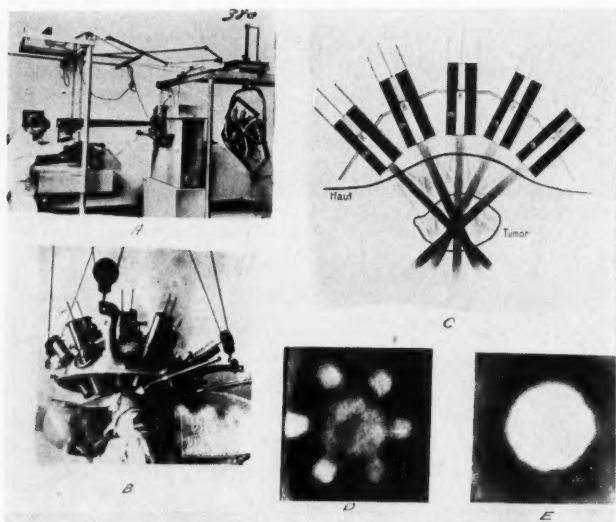


Fig. 35. Teleo-Radium therapy apparatus. Radium bomb.

II.

SOME NOTES ON THE ETIOLOGY AND TREATMENT OF TINNITUS AURIUM.*

J. A. BABBITT, M. D.,

PHILADELPHIA.

The assertion was ventured, in a recent published study, that the two more nearly unsolved problems in otolaryngology were chronic suppurative otitis media and progressive deafness. Tinnitus aurium is an integral phase in each, its problem is a major one and it is timely to collaborate and balance the results of study in this and other countries. In Jones and Knudsen's¹ recent statistics, tinnitus was present in 59 per cent of 340 cases of hearing impairment analyzed, with a higher percentage in conductive than perceptive cases. To this conductive group must be added its presence in varying degree in suppurative cases.

Most painstaking work has been put upon the investigation of tinnitus, particularly in the last two decades. Such contributions are those of Lake² on operative measures and relations of blood pressure; Alford,³ Parish,⁴ Williams⁵ and Woakes⁶ on mental analogies and hallucinations; Bryant⁷ and Woakes⁶ on etiology and classification; Jones and Knudsen¹ on analysis, bombardment and masking methods in treatment; Forster⁸ and Ruskin⁹ on sympathetic association; Gray,¹⁰ Harris,¹¹ Lothrop,¹² Pohlman¹³ and Mackenzie¹⁴ on general nature and theory; Scal¹⁵ and Drury¹⁶ on endocrines; Mollison¹⁷ and Sturm¹⁸ on internal medication; Kirk,¹⁹ Tansey²⁰ and Wright²¹ on dental factors; Araya, Worms, Moothi and Rateau²² on electro-anesthetic currents; Bunch,²³ Jones, Knudsen, Josephson,²⁴ Macfarlan²⁵ and Wegel²⁶ on precisional measurements; Friedlander and Sommer²⁷ on climacteric influence; Kinney,²⁸ roentgen therapy; Felderman and Dyson²⁹ on spinal injections of glucose; Benito,³⁰ Covilli-Fagioli,³¹ Hays,³² Linn,³³ Lieri³⁴ and Zimmerman³⁵ on specialized treatments.

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, New York City, January 9, 1932.

Valuable to this has been the recent clarification of middle ear and mastoid pathology by Kopetzky, Almour³⁶ and others. The list is far from complete but will serve to indicate the scope of work and authors will be freely quoted. Acknowledgment is made of the service of Doctors Sink and Wolff in search of literature.

Practical results have been far from commensurate with effort expended. Even exact measurements of pitch and intensity, valuable from scientific standpoint, scarcely suggest the cure for tinnitus. It is the purpose here to review briefly the fundamental etiology and accepted methods of treatment as an introduction to the presentation of an interesting problem case for discussion and suggestion.

Nature and Origin of Tinnitus.—Tinnitus aurium is not an entity save as an expression recognized in acoustic cerebral centers of simple or compound stimulation of peripheral acoustic apparatus. It is accepted that to reach such registration it must be either cochlear in origin or transmitted in vibration to the cochlea, from circulation, middle ear irregularity, muscular tension, sympathetic or other adjacent influence. The complexity of influences forming the syndrome of tinnitus renders it impossible to isolate distinctive pathology. Abnormalities, functional or organic, in the following fields appear, however, definitely causal: 1, eustachian tube; 2, tympanic cavity; 3, external auditory canal; 4, labyrinthine circulation; 5, neurolabyrinthine apparatus.

Eustachian Tube.—This presupposes pathology in the walls with stenosis somewhere in its course, though voluntary and involuntary closure may be brought about by muscular action. Tinnitus, audible to both patient and outside observer, is due to this eustachian tube origin, occurring usually in the form of a subdued click.

Tympanic Cavity.—In reference to intratympanic pathology, Ballenger's physiologic law deserves restatement: "Anything that interferes with the normal tension existing between the membrana tympani, ossicles and contents of the oval window will cause tinnitus and deafness." Such would include pressure of fluid in the middle ear, serous or suppurative, secondary atrophic changes, sclerotic and adhesive processes, particularly in the

ossicular chain, ankylosis of the footplate of the stapes. Even simple inflammatory thickening of mucous membrane in tube and tympanic cavity could disturb this normal tension. Patients refer to the superficial, rather than deep type of tinnitus in chronic catarrhal otitis media. The symptom complex of chronic purulent otitis media is otorrhea, deafness and tinnitus.

External Canal.—Obstructive irritation here includes stenosis and pressure from exostosis, impacted cerumen, foreign bodies, furunculosis, eczematous thickening, syphilitic condyloma. Perhaps Arnold's branch of the tenth and the disturbance of the auriculotemporal may have some indirect significance.

Labyrinthine Circulation.—Hyper- and hypo-arterial tension, with resultant anemia and hyperemia, produce persistent types of pulsating tinnitus and venous congestion, extending into the intracranial sinuses, may cause the chattering, chirping or tree rustling sounds. Both olfactory hallucinations and tinnitus appear in the anemia following hemorrhage, perhaps analogous to the pulmonary hemic murmur. Alcohol and tobacco in excess, by withdrawing vessel inhibition, may produce a type of venous congestion tinnitus.

Neurolabyrinthine Apparatus.—This is basic in cerebral interpretation of tinnitus sounds—and becomes corollary to all other factors in etiology.

Whether conductive or receptive, functional or organic, it must primarily pass through the organ of Corti. If originating in the cochlea, it seems reasonable to assume some cochlear degeneration. In certain individuals this accords with certain constant variations in the audiogram curves—variations of the type presented by Fowler³⁷ before the Otological Society; sometimes pitch seems in agreement but not too frequently. Bryant⁷ assumed that the causes of otic tinnitus were identical with the causes of deafness. Where tinnitus and deafness periods coincide in the cochlear turn, it is probable that the deafness is much dependent upon tinnitus, masking tone perception. Tinnitus relation to the islands of acoustic gap in the cochlea remains unsolved. It will probably be accepted that the tinnitus sound is not the objective sound carried to the cochlea.

It is assumed that the majority of us believe that deafness and its accompanying tinnitus must have their primary origin in the cochlear path, and this is not out of harmony with Gray's,¹⁰ Wrightson's³⁸ and Pohlman's¹³ views on the transmission of sound and the functional part played by the round window. Understimulation or activation of an inadequate number of nerve fibers and hair cells in the organ of Corti produces deafness; overstimulation should produce tinnitus which in certain tonal pitch increases deafness. The important point is to determine how in tympanic irregularity, the net result is transmitted to the cochlea as vibratory irritant.

Perhaps the suggestion of stimulated tonic vibration of the antagonistic intratympanic muscles, stapedius and tensor tympani will satisfy some cases. What, however, about the tinnitus occurring with no demonstrable deafness—and the aggravated tinnitus of otosclerosis, whose tympanic membrane may be so normal and in which promontory even redness and stapes fixation may be difficult to diagnose?

There seems rather a curious analogy in tinnitus to what is termed atypical neuralgia—clearly defined in itself, yet indefinite as to source, conditioned in large degree to systemic physical states, yet only remotely referable to demonstrable focus of infection.

Enthusiasts upon the subject of endocrine imbalance, sympathetic vasomotor system, cumulative results of dental malposition and distal influence of focal toxemia will claim an equal or more disturbing relation than those of the primary list cited. Added weight to the endocrine picture might be given by the newer views of thyroid, epinephrine and pituitary contribution to lowered vitality or resistance, as found in the so-called status lymphaticus—but there fails to appear any definite correspondence with such state and tinnitus aurium. The vasomotor influence is, of course, important. Harris,¹¹ quoting Reik's³⁹ theory from animal experimentation that increase in intratympanic pressure causes vasomotor dilatation resulting in a lowered blood pressure—and lowered blood pressure had been demonstrated in most persons suffering from tinnitus—gives the opinion: "Subjective auditory sensations generally result from vasodilatory changes

in the vasomotor systems of the middle or internal ear, and may be caused by any case of irritation which will affect the vasomotor nerves controlling these vessels; consequently, that stimulation of the vasomotor of the ear produces a depression effect, small vessels become dilated and the abnormally large amount of blood coursing through these give rise to new sounds, to which the auditory nerve is unaccustomed." It is hoped that this reported uniformity of low blood pressure will be discussed. Experience with a considerable number of such cases has given the impression of a reverse percentage.

The influence of malposition of the jaws in causing deafness and incidental tinnitus has been well presented by Wright of Pittsburgh. He attributes this loss to the abnormal relationships of the mandible with the maxillæ, producing habitual posterior displacement of the mandibular condyles. Apparently the results from three causes—first, loss of some or all of the teeth; second, malposed teeth, permitting malocclusion; third, extensive abrasion—produce a closed bite. The important points from the otolaryngologic standpoint are, first, the compression on the anterior wall of the external auditory meatus, which comprises the principal part of the glenoid fossa in which the condyle of the mandible articulates; and second, the proximity to this glenoid fossa of the Glasserian fissure lodging the processus gracilis of the malleus and transmitting the tympanic branch of the internal maxillary artery, and the chorda tympani passing through the canal of Hugier on the outer side of the eustachian tube. This tympanic plate, in front of the petrous portion and separating the external auditory canal from the glenoid fossa, is in turn separated from the articular portion of the fossa by the Glasserian fissure which opens into the tympanic cavity. The inferior roughened margin of the tympanic plate lies around the base of the styloid process. As shown by Wright, this plate is inclined inward and forward, while the condyle is inclined inward and backward, which places the inner end of the condyle, in some normal cases, as far back as the Glasserian fissure or across it. It is logical to assume that both irritation and vibration from the jaw and neck muscles may be carried through to the middle ear and perhaps conveyed on to the cochlea. The occasional open hanging

jaw in the listening attitude indicates that the hearing is improved by release of pressure about the joint, and change has been noted in the patency of the pharyngeal end of the tube when the bite has been artificially corrected. Experience of important cases in the wearing of artificial blocks to accomplish this has not demonstrated relief to deafness or tinnitus.

The question of focal toxic background is naturally linked to all pathology in the upper respiratory area, including the middle and internal ear. There is a certain analogy in Emerson's⁴⁰ discussion of progressive deafness, which seems germane to the question of conductive and perceptive elements in tinnitus. There is an early stage in tubal involvement and stenosis, associated with retraction of the tympanic membrane which is marked by hyperplasia or hypertrophy of tubal structure—conductive deafness predominates and local treatment seems helpful. After a period of years this is succeeded by atrophy, opening of tubal passage and often relaxation of tympanic membrane. Emerson believes that the focal toxic elements have been effective from the beginning in producing perceptive apparatus change while this has been marked by conductive symptomatology. This seems logical, and the thought persists that probably tinnitus, apparently associated with the middle ear to the exclusion of the labyrinth, has been occupied with the same process.

Much discussion and theorizing have been devoted to the question of central or peripheral seat of tinnitus. There seems little doubt that tinnitus, originating in the cochlear region, after a certain period, perhaps empirically stated as from three to four years, becomes a central memory tinnitus. Frazier's case, cited by Jones,¹ of later return after section of the eighth nerve and cases returning after surgical destruction of the cochlea bear this out. This invades the bewildering field of psychiatry and hallucination, and the schizophrenic and manic depressive almost require a hearing in this otolaryngologic realm. Time does not allow discussion of the picturesque views of Woakes in working out the aural influence upon leaders of some of the great world movements, whose ascetic lives, self-imposed privations and exposure have provided the background for infections of the otolaryngologic tract, precursors to otitis media, deafness, vertigo

and tinnitus. Such tinnitus is often the talkative, chattering type. The interpretation of this by a fanatic mind as articulate sound or spiritual voice would be natural.

Classification of tinnitus may be in very simple grouping, such as with and without hearing defect, tonal or mixed sound (noise), conductive or perceptive origin, degree of intensity, continuity, seasonal relation, occupational, etc., or very elaborate, as the grouping early arranged by Sohier Bryant and based upon etiology, with subjective and objective divisions.

Therapeutic Measures.—The views of various investigators have been woven into this presentation by allusion and comparison. The one definite impression, both from experience and review of the literature, is that all methods advocated have had a modicum of success. Some cases were apparently cured, some cases here and there relieved for a time but more often relapsed. Often the secondary state of comfort would appear to be a sort of auto-suggestion tolerance, sometimes a fatigue reaction to treatment.

Under treatment, and of course following exhaustive study of the patient and his upper respiratory tract, must be considered systemic treatment, specific internal medication, local surgery, X-ray, galvanic and other electrical attacks, bombardment and masking measures, endocrine regulation, injection of posterior palatine for sphenopalatine control, dental correction in malocclusion and perhaps, least important of all, routine tubal treatment.

All possible information must be elicited as to specific disease, tuberculosis, exanthematous complications, alcohol, tobacco, recent administration of drugs, climatic and living conditions, hereditary sequence in otosclerosis, with complete laboratory study, eyegrounds and search for focal infection. Hearing should be carefully studied, as the patient with tinnitus may not have yet recognized a hearing fault, particularly unilateral. The single important axiom is to find and remove the cause before it becomes irremediable.

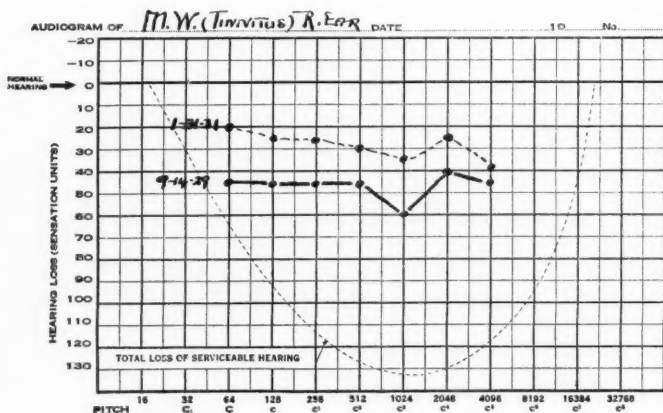
NOTES ON ROUTINE MEASURES.

1. *Systemic Reconstruction.*—The work of an earnest group of otolaryngologists working on the question of nutritional disaster

in its relation to the upper respiratory tract has been studied and deserves serious consideration in this connection. This should not be confounded in any way with the organized and expensive dietary "schools" now in vogue. Jarvis⁴¹ has demonstrated that diagnostic help in the mucous membrane—the whole problem of balanced diet, acid and alkali tolerance, vitamin influence—has a therapeutic bearing on the factors behind tinnitus and progressive deafness. Cases of tinnitus have shown marked response; migraine and atypical neuralgia negative to focal study have apparently cleared up under nutritional reorganization.

2. *Routine Treatment and Minor Surgery.*—When tinnitus occurs from tubal congestion and beginning hyperplasia, routine intratubal measures, begun early, may be helpful, but protracted treatment under other conditions has only psychic apology. Removal of focal elements in nasopharyngeal tract and teeth, obstruction in external canal and nares are justified.

In connection with the writer's procedure in attic drainage for chronic middle ear infection, some definite cases of relief from tinnitus have occurred, coincident with improved hearing, but not complete elimination. It has proved a worth while therapeutic measure.



Audiogram before and after attic operation with improved hearing and reduction of tinnitus. (M. W.)

3. *Specific Medication.*—English otologists have recommended dilute hydrobromic acid, often combined with potassium iodide. Scal¹⁵ has reported favorable averages in the use of thyroid extract, if unsuccessful substituting adrenalin substance. He used successfully ovarian extract in several menstrual cases. Donovan's solution and the use of sodium bicarbonate in large doses for three days, and then followed by nitrohydrochloric acid for several more, as advocated by members of the nutrition group in connection with dietary regulation, has given periodic relief. Blood pressure changes give their own medical indication. Kerekes¹² gave sodium nitrite subcutaneously in tinnitus aurium, reporting 12.7 per cent of cases cured and 63.6 per cent improved. Sacher¹³ used mercury, and Sturm advocated the local use of atropin solution, using on the tympanic membrane if intact, through perforation if present.

4. *Mechanical and Electrical Measures.*—Jones and Knudsen¹ have devised two instruments, one for bombardment of the cochlea, suggested by animal experiments of Silverman, Wittmaack and Yaskin. The object is to desensitize the cochlea in certain tonal regions, after precisely determining pitch and loudness. This is a fatigue process and is not lasting. The other, a harmonic generator, producing a complex spectrum of tones, helpful in sleep. In patients with conductive impairment, this blankets and gives relative hearing. It is reasonable to assume that some adequate and comfortable noise apparatus, adjustable in pitch and intensity, may be soon devised to meet the cases of tinnitus which have well nigh reached the suicidal stage. X-ray treatment and the various electrical currents, particularly galvanism, have hardly reached a definitely successful stage.

5. *Major Surgery.*—It is significant that such major procedures as obliteration of the cochlea (Lake) or section of the eighth nerve have been discarded, as both dangerous and untrustworthy. A nearly major procedure in connection with middle ear surgery will be presented.

Special Problem Case.—It was the intention to present with this paper some pathologic studies of tinnitus ears, and group studies of patients, but definitely related necropsy material was not secured, and routine cases proved too variable for adequate

averages. In their place, this interesting problem case of severe tinnitus and measures taken for relief is presented.

Personal History.—The patient, Mr. B. B., single man, 27 years of age, was referred by Dr. Dorrance to the University Clinic for further study in May, 1931. His tinnitus commenced with a bad head cold twelve years ago—first in left, then in right a year later, and now dominant on right side. His reports as to its character, relation to exertion, breathing and swallowing pointed to tubal involvement. The tonsils had been removed nine years ago and the patient had been under careful routine nose, throat and ear treatment three times a week for the past four years. After varied specialists had examined him, the patient was sent to Dr. Kirk of the Dental School, under whose advice a jaw muscle transplant was done by Dr. Ivy. (Masseter attachment transposed forward to draw on condyle.) Silver bite blocks were worn for one year without result, teeth had been overhauled and some extraction done. Patient sleeps badly, intensely nervous, tinnitus has at times become discordant, suggesting diplacusis, but presents no suggestion of either hallucination or malingering.

Otolaryngologic Study.—No nasopharyngeal focus of infection was found. Intranasal examination satisfactory. Tympanic membranes somewhat retracted and fibrous, otherwise negative. Hearing practically normal to audiometer and tuning fork tests. Rinne positive, no lateralization in Weber test, bone conduction normal.

Bárány Studies (Dr. Winston).—"The vestibular examination showed loss of function of the vertical and horizontal semicircular canals of the right and likewise of the left vertical, with considerable loss in the left horizontal. These findings were considered due to a toxic labyrinthitis involving the vestibular portion of both ears, rather than an intracranial lesion."

Medical, neurologic and X-ray studies scarcely add value to this report.

In routine treatment various intratubal measures were tried without success. Internally dilute hydrobromic acid and locally liquor atropin, as recommended by English authors, were ineffective. Iodides, blood pressure medication and endocrine extracts also failed. It was decided to try the Felderman and Dyson spinal injection of glucose on the theory of changing the spinal

fluid pressure in the third ventricle and ductus endolymphaticus, thereby relieving intralabyrinthine pressure. This was done at the University Hospital, under direction of Dr. Hitzrot, but failed to give relief. Laboratory tests on the spinal fluid showed 4 cells per c.mm., 1 unit of protein and a normal sugar content. Colloidal gold curve and Wassermann were negative.

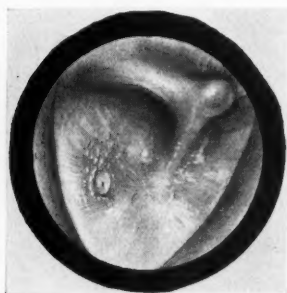
As a final measure, the tympanic membrane was incised, near the annulus, in the external half of the membrane, to relieve tension. This procedure was the first to give any indication of relief and for a short period only.

This repeated failure began to evidence a memory tinnitus. Then an interesting thing happened. After a few weeks' vacation from treatment in the New Jersey woods, the patient returned and reported discovery of a method for relief. He had found that by working a finger deeply into the external canal of the more affected side and then drawing it slightly out, producing suction, his tinnitus was relieved. An attempt was made to imitate this by insertion of rubber and other plugs and exhaustion of air behind. This was successful, but the procedure caused much external inflammation after 24 hours and was abandoned.

Therefore, on December 11th, at the Lankenau Hospital, as the patient was desperate and ready to sacrifice hearing for relief, the following rather extreme measure was tried. Under careful antisepsis, an elliptic incision was made in the right tympanic membrane, starting low on the external border, following up just inside the annular margin, across at the level of Shrapnel's membrane to base of the long process of the malleus, then down along its outer side to below the umbo. The flap was turned down, exposing the tympanic cavity and ossicles. The stapedius muscle was severed, and with curved tenotome passed beneath the malleus, and reinforced by an incision anterior to it, the tendon of the tensor tympani was cut and other adhesions removed. With right angled finder, the attic area was swept clear of adhesions and reachable ligaments. The malleolar tip was raised outwardly and the flap restored. There was temporary vertigo, but immediately after the operation the tinnitus absolutely departed and remained practically absent for about a week. It has returned periodically and in minor degree, but can be relieved by tympanic



Tympanic membrane shortly after first operation.
August, 1931. (B. B.)

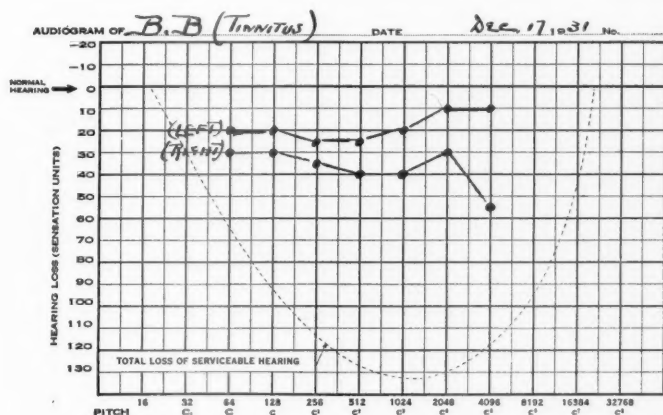


Tympanic membrane practically healed, Jan. 29, 1932. (B. B.)

manipulation and clearing of granulations. There has been no infective sequence.

This separation of intratympanic musculature and freeing of intratympanic space had at least proved that his tinnitus had not become a memory tinnitus in the higher acoustic centers, and though some little further manipulation may be required to keep this elastic the solution of his trouble has perhaps been reached.

The purpose of this presentation was to testify to the inadequacy of conventional measures in this and other cases and to open the discussion as to the value of possible research in this situation offered by the stapedius and tensor tympani muscles.



Audiogram of special reported case, taken one week after operation. (B. B.)

This particular case, which was thoroughly and carefully done, demonstrated immediate relief. The judgment of its research value is left to the discussion of this meeting. But three simple conclusions will be offered:

1. The syndrome of tinnitus aurium is too complex to justify any conventional routine treatment.
2. In the absence of cochlear degeneration or loss of function in the eighth nerve, the tympanic cavity offers a promising surgical field for the relief of tinnitus.
3. The assumption of memory tinnitus without demonstrable labyrinthine or eighth nerve involvement is unwarranted.

1912 SPRUCE STREET.

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III.

EARLY INTRINSIC CANCER OF THE LARYNX, DIAGNOSIS AND TREATMENT: OBSERVATIONS ON LARYNGOFISSURE AS A METHOD OF TREATMENT IN A SERIES OF CASES.*

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PHILADELPHIA.

"The accurate study of laryngeal cancer has been possible only since 1855, when Manuel Garcia invented the laryngoscope."

Today we have the improved and perfected laryngoscope after the model of Garcia, and by its use laryngologists should be able to detect a lesion in the larynx and, as Dr. Delavan has so well said, "by subconscious reasoning based upon long experience of many closely observed cases," make a correct diagnosis of *early* cancer of the larynx. Every otolaryngologist who is especially interested in the larynx will verify, however, the fact that today the diagnosis is rarely made early by the rank and file of physicians who first see these patients. The principal reason for this is that the great majority of physicians do not take the time to acquire the technic required for accurate visualization of the larynx. In the rush of routine work a hoarse patient, after an imperfect visualization of the larynx, is given a gargle, or, worse still, a local application of silver nitrate which will produce a burn and mask the appearance of the disease in the larynx that might lead to proper diagnosis. The patient is told that he has a chronic laryngitis and is treated for this until the cancer is far advanced.

As a means of accurate diagnosis in a laryngeal lesion that has been studied properly by mirror examination and the routine diagnostic studies which are so well known that they need not be repeated, we have a method of examination which should be used supplementary to the mirror as the final step in differential diag-

*Read before the American Academy of Ophthalmology and Otolaryngology, September 18, 1931, at French Lick, Indiana.

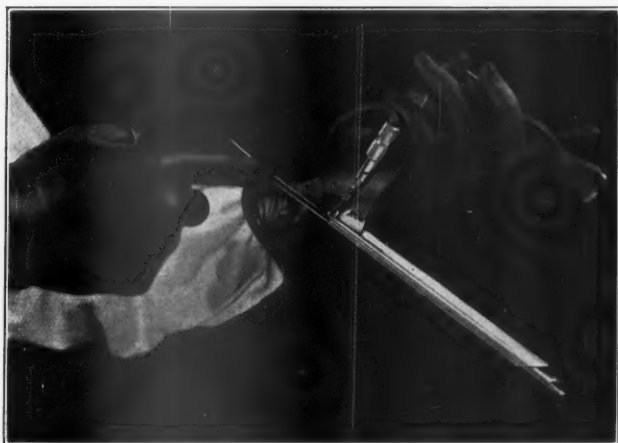


Fig. 1. The Jackson anterior commissure laryngoscope, and basket tissue forceps, in correct position for removal of specimens of tissue from the larynx. With the patient in dorsal recumbency under local anesthesia, tissue may be removed from any portion of the larynx desired.

nosis, namely, direct laryngoscopy and biopsy by the method of Chevalier Jackson. By this method every part of the larynx can be seen and tissue removed accurately from any location. (Fig. 1.)

The length of this paper will not permit a full discussion of intrinsic cancer of the larynx, but every physician, and more particularly every laryngologist, should read carefully "Cancer of the Larynx," by Sir St. Clair Thomson, the dean of British laryngology, and his very able associate, Mr. Lionel Colledge. In large part, due to the teaching and practice of Sir St. Clair Thomson in the British Isles and of Dr. Chevalier Jackson in the United States, American laryngologists are realizing that intrinsic cancer of the larynx, if recognized early, is, in the majority of cases, in such a location that it can be removed surgically without the removal of the larynx, with a very high percentage of cure. Early diagnosis, then, is "the pearl of great price" in the cure of intrinsic cancer of the larynx. In the cases that are not amenable to

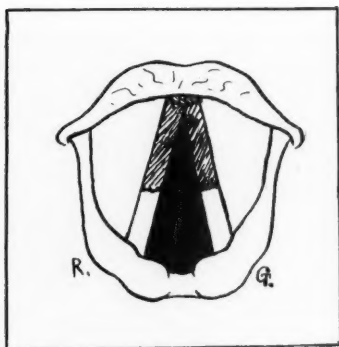


Fig. 2. Schematic drawing of the larynx. The shaded area represents the "anterior intrinsic area." Cancer limited to this area, on either side or the anterior commissure is amenable to cure by laryngofissure.

laryngofissure early diagnosis is also of very great importance in order that laryngectomy may be done before metastasis takes place. We may speak, then, of intrinsic cancer of the larynx as being early if a diagnosis can be made before metastasis has occurred. Intrinsic cancer of the larynx occurs most often in the "anterior-intrinsic area" (Jackson). This area includes the anterior two-thirds of the true vocal cords, the adjacent portions of the ventricles of the larynx, and the anterior commissure. (Fig. 2.)

A growth occurring in this area is amenable to removal by laryngofissure because of the tendency to late metastasis due to the peculiar lymphatic arrangement. (Cuneo.) (Fig. 3.) Also, this is the area in which the lesion is most apt to be overlooked in mirror examination because of the difficulty in many patients in getting a clear view with the mirror of the anterior intrinsic area.

Diagnosis: Chronic hoarseness is a constant symptom in practically every case of intrinsic cancer of the larynx. A growth involving the ventricular band, according to Sir St. Clair Thomson, may produce only slight impairment of the voice. There is, however, much more frequently a sticking sensation, or actual pain, which should lead to careful examination and a correct diagnosis

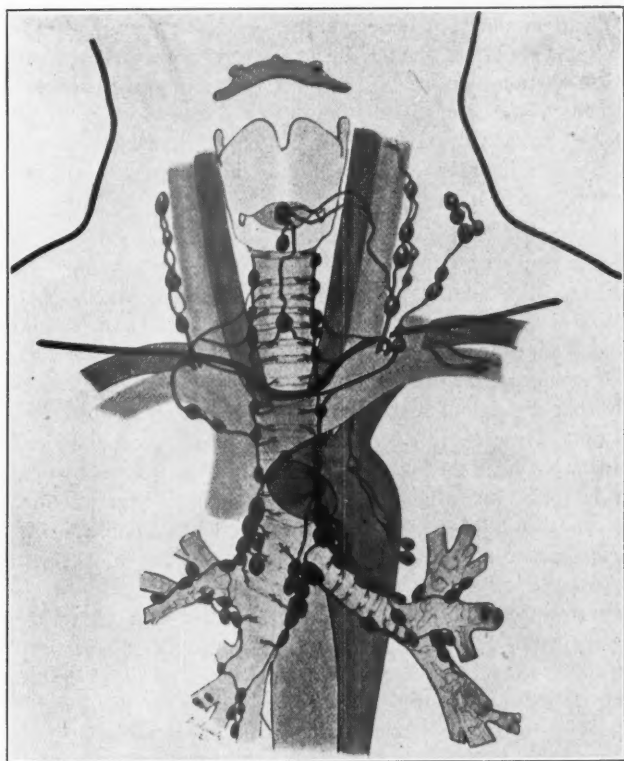


Fig. 3. Lymphatic glands of the larynx, trachea and bronchi.
The anterior intrinsic area drains through the pretracheal group.
(Professor Dr. M. Hajek, after Thompson and College, London.)

of early involvement. This applies also to lesions in the deep subglottic area. The treatment of involvement in the subglottic area and ventricular bands is not so successful by laryngofissure unless the lesion is of very limited extent.

The routine diagnostic procedure which has been so often repeated in teaching and so infrequently carried out in practice in the individual case, should be, first, careful examination and routine study of the larynx with a mirror of variations from normal

in color of mucous membrane, in motility, and in the contour of the larynx should be made carefully. Second, the general examination of the patient should include a careful history, a general physical examination, careful roentgen-ray study of the neck and chest, blood and serological studies, in order that we may determine the relation of general or systemic disease to the local lesion in the larynx. The final step in differential diagnosis should be direct laryngoscopy and biopsy.

Biopsy: There have been many objections to biopsy, the first one being the fallibility of the pathologist's report. The report of the pathologist should always be taken in conjunction with the clinical findings in the case. If it is a question of the interpretation of the histologic picture, any pathologist will welcome a consultation with a peer. The complaint has been made that the pathologist pronounces the condition cancer when it is not. That has not been my experience. Another objection to biopsy has been the possible production of metastasis by biopsy. This is very well refuted, however, by the fact that we can get a report from a fixed specimen, in a period of 48 hours, certainly before there could be opportunity for metastasis to occur. All our pathologists agree that there have been no bad results from this procedure when properly performed. Biopsy by direct laryngoscopy gives a certainty of location and accuracy of removal that can be obtained in no other way. I have never regretted doing a biopsy for a differential diagnosis, but I have regretted very much my inability to get permission to do a biopsy until the lesion was far advanced.

Early biopsy will frequently reveal a precancerous condition. Operative removal in the stage when the lesion is precancerous will require a much less radical procedure to effect a cure than after the lesion is so far advanced that its nature is perfectly evident on mirror examination. In a recent case the author found both tuberculosis and cancer in the same microscopic section. (Figs. 4, 5 and 6.)

Pathology: McKenty gives the relative frequency of the different forms of carcinoma of the larynx as squamous carcinoma, 96 per cent; basal cell carcinoma, 2 per cent; papillary carcinoma, 1 per cent; adenocarcinoma, 1 per cent. The careful grading of

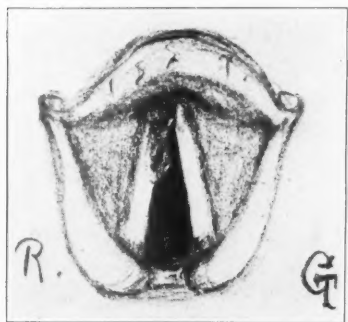


Fig. 4. Sketch of mirror image of the larynx showing irregular ulcerating mass of tissue occupying the anterior third of the right cord. Direct examination showed the lesion to extend across the anterior commissure to the under surface of the left cord. The other portions of the larynx were free from involvement.

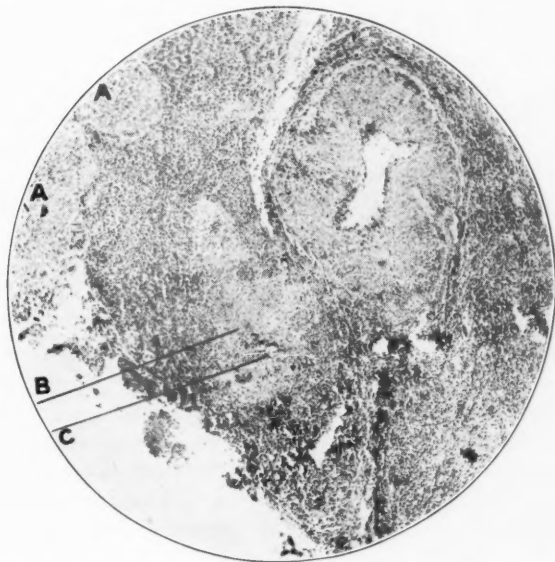


Fig. 5. Low power microscopic section from biopsy specimen showing areas of cancer and tuberculosis: A, A, cancer; B, tuberculosis; C, giant cell.

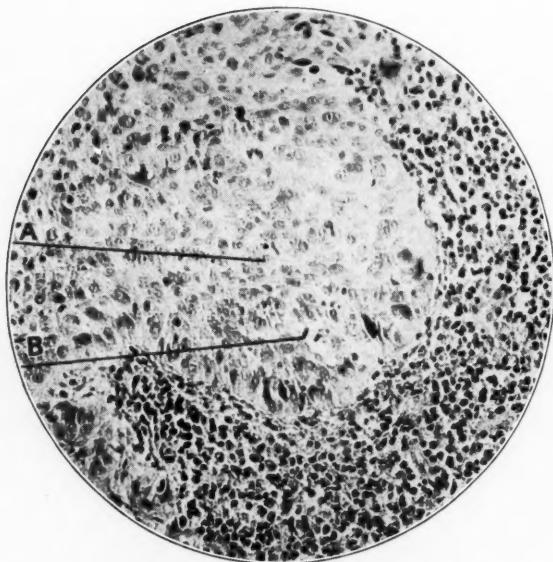


Fig. 6. High power view of cancer area (A) showing mitotic figure (B). Dr. E. A. Case.

the cancer, according to Broder's classification, is advocated by Gordon New as a means of determining the degree of malignancy. Not all pathologists, however, follow this method of grading, and clinically we must depend more upon the location and the extent of the lesion as found on mirror and direct examination than on the grading of the histologic characters. It would seem advisable to remove a growth at laryngofissure, even if of the highest grade of malignancy, if it was in a favorable location and of limited extent.

It is generally recognized that cancer of the larynx is not highly malignant in type, and if the cases are closely followed after laryngofissure, a laryngectomy can be done if recurrence takes place. This gives the patient a chance to keep his larynx and no harm results. (See case 25 in series.)

A series of 30 cases of laryngofissure for early intrinsic cancer of the larynx from the bronchoscopic clinics of the University

of Pennsylvania, operated upon by the author, is presented in tabulation. (See table.) There was no operative mortality in the series. In twenty-five cases over one year has elapsed since the operation. There was no operative mortality in the series. In twenty-five cases over one year has elapsed since the operation. There have been two recurrences in this group. One recurrence occurred nine months after laryngofissure; the patient refused laryngectomy and died at the end of one year after operation. The second case had a recurrence at one year and five months after partial laryngectomy, and a second recurrence ten months later, which will require laryngectomy. Five cases in the series are recent, under one year, have no recurrence, but are not considered in the percentage estimate. The longest duration of cure is seven years; eight cases are of over four years' duration.

The technic of laryngofissure used in the series follows very closely that of Chevalier Jackson. Thyrochondrotomy of Sir St. Clair Thomson was employed in several cases.

In the first half of the series general anesthesia, intratracheal ether, was used. At the present time local anesthesia only is employed. Technic of laryngofissure: The patient is carefully prepared after a diagnosis of cancer has been made by biopsy. Blood chemistry, kidney function tests and general examination by a competent internist are carried out. Preliminary morphin and atropin is not administered unless the patient is very apprehensive or of a particularly nervous temperament. The patient is placed in dorsal recumbency with a sand pillow beneath the shoulders with the head slightly lowered. One-half per cent novocain is infiltrated into the front of the neck in the median line from the hyoid bone to the suprasternal notch.

The front of the larynx, cricoid and upper tracheal rings are exposed. Great care is used not to skeletonize the larynx or to injure the external perichondrium of the thyroid cartilage. Bleeding points are ligated. The larynx is then opened by incising the cricothyroid membrane. Through this incision is introduced a small bronchoscopic sponge, saturated with 10 per cent cocain. The interior of the larynx is swabbed with this solution, sufficient being applied to produce anesthesia. If the cancer is limited to one cord and does not involve the anterior commissure the

THYROTOMY OR LARYNGOFISSURE IN THE CURE OF EARLY INTRINSIC CANCER OF THE LARYNX.

CASE SERIES FROM UNIVERSITY OF PENNSYLVANIA BRONCHOSCOPIC CLINICS.

TABULATION OF CASES.

Series No.	Age and Sex Occupation	Duration of Symptoms	Location of Lesion	Date of Biopsy	Thyrotomy date Anesthesia	Convalescence Complications	Recurrence Metastasis	Result	Period of cure to Sept. 1931
1.	Male, 64 yrs. Plumber	Hoarseness 6 months	Anterior 3rd right cord	Squamous epithelioma Aug. 1, 1924	Aug. 5, 1924 Intra-tracheal ether	2 weeks granuloma	None	Voice clear Airway normal	7 years and 1 month
2.	Male, 33 yrs. Broker	Hoarseness 3 years	Entire right cord and ventricle	Inflammatory epithelioma May 7, 1925	May 29, 1925 Intra-tracheal ether	3 weeks granuloma	None	Voice hoarse Airway normal	6 years and 4 months
3.	Male, 60 yrs. Brick worker	Hoarseness 18 months	Anterior 3rd L. commissure	Squamous epithelioma Oct. 11, 1926	Oct. 15, 1926 Intra-tracheal ether	3 weeks	None	Voice hoarse Airway normal	4 years and 11 months
4.	Male, 54 yrs. Salesman	Hoarseness 1 year	Anterior 3rd left cord	Squamous epithelioma Oct. 11, 1926	Oct. 15, 1926 Loc. novocaine and cocaine	2 weeks	None	Voice clear Airway normal	4 years and 11 months
5.	Male, 56 yrs. Univ. Prof.	Hoarseness 4 months	Anterior commissure	Basal cell carcinoma Dec. 9, 1926	Dec. 11, 1926 Loc. novocaine	3 weeks granuloma	None	Hoarse voice Airway normal	4 years and 9 months
6.	Male, 70 yrs. Ins. Agent	Hoarseness 4 months	Anterior commissure	Squamous carcinoma March 8, 1927	March 15, 1927 Intra-tracheal ether	3 weeks	None	Hoarse voice Airway normal	4 years and 6 months
7.	Male, 65 yrs. Salesman	Hoarseness 8 months	Anterior commissure	Squamous epithelioma April 11, 1927	April 14, 1927 Intra-tracheal ether	2 months slight perichondritis	None	Voice good Airway free	4 years and 5 months
8.	Male, 44 yrs. Automobile Mechanic	Hoarseness 9 months	Mid-third left cord	Squamous carcinoma May 3, 1927	May 6, 1927 Intra-tracheal ether	4 weeks (3) granuloma	None	Good voice Airway normal	4 years and 4 months
9.	Male, 54 yrs. Civil Engineer	Hoarseness 8 months	Mid-third left cord	Papilloma beg. epithel.	Nov. 4, 1927 Intra-tracheal	4 weeks	None	Good voice Airway normal	3 years and 10 months

10.	Fem., 35 yrs. Housewife	Hoarseness 8 months	Anterior 3rd right cord	Papilloma beg. epithel. March 27, 1928	April 3, 1928 Intra-tracheal ether	4 weeks granuloma	None	Good voice Airway normal	3 years and 5 months
11.	Male, 68 yrs. Barber	Hoarseness 8 months	Mid-third left cord	Epithelioma April 24, 1928	April 28, 1928 Loc. Nov. & Coc.	4 weeks	None	Good voice Airway normal	3 years and 5 months
12.	Male, 57 yrs. Salesman	Hoarseness 2 years (intermittent)	Anterior 3rd right cord	Papilloma and early malig. April 20, 1928	May 3, 1928 Loc. Novocaine	3 weeks granuloma	None	Good voice Airway normal	3 years and 4 months
13.	Male, 54 yrs. Salesman	Hoarseness 6 months	Mid-third right cord	Epithelioma May 10, 1928	May 12, 1928 Loc. Nov. & Coc.	2 weeks granuloma	None	Voice clear Airway normal	3 years and 4 months
14.	Male, 57 yrs. R. R. Eng.	Hoarseness 7 months	Anterior two-thirds left cord	Epithelioma June 4, 1928	June 12, 1928 Loc. Novocaine	2 weeks granuloma	None	Voice clear Airway normal	3 years and 3 months
15.	Fem., 57 yrs. Housewife	Hoarseness 1 year	Anterior two-thirds right cord	Epithelioma Sept. 10, 1928	Sept. 13, 1928 Intra-tracheal ether	4 weeks	None	Voice good Airway normal	3 years
16.	Male, 53 yrs. Carpenter	Hoarseness 5 months	Anterior commissure both cords	Inflammatory epith. change Oct. 30, 1928	Nov. 13, 1928 Loc. novocaine	4 weeks perichondritis	None	Voice good Airway free	2 years and 10 months
17.	Male, 63 yrs. Huckster	Hoarseness 5 months	Anterior 3rd left cord	Squamous cell carcinoma April 1, 1929	May 8, 1929 Loc. Nov. & Coc.	3 weeks granuloma	None	Voice good Airway clear	2 years and 4 months
18.	Male, 58 yrs. Minister	Hoarseness 8 months	Mid-third left cord T. B. pulm.	Squamous carcinoma May 15, 1929	May 18, 1929 Loc. Nov. & Coc.	3 months perichondritis	None	Voice good Airway free	2 years and 4 months
19.	Male, 64 yrs. R. R. Cond.	Hoarseness 11 months	Ant. commis- sure and left cord	Squamous cell carcinoma June 22, 1929	June 18, 1929 Loc. Nov. & Coc.	Perichon- dritis	Recurrence at 9 months	Atresia of larynx Death	2 years and 3 months
20.	Male, 63 yrs. Salesman	Hoarseness 1 year	Anterior two-thirds left cord	Squamous cell carcinoma June 22, 1929	June 26, 1929 Loc. Nov. & Coc.	2 weeks	None	Voice good Airway normal	2 years and 3 months

THYROTOMY OR LARYNGOFISSURE IN THE CURE OF EARLY INTRINSIC CANCER OF THE LARYNX.—(Cont.)

Series No.	Age and Sex Occupation	Duration of Symptoms	Location of Lesion	Date of Biopsy	Thyrotomy date Anesthesia	Convalescence Complications	Recurrence Metastasis	Result	Period of cure to Sept. 1931
21.	Fem., 52 yrs. Housewife	Hoarseness 18 months (intermittent)	Anterior commissure right cord	Papilloma beg. cancer July 30, 1929	Aug. 26, 1929 Loc. Nov. & Coc.	10 days	None	Voice good Airway normal	2 years and 1 month
22.	Male, 48 yrs. Prof. Golfer	Hoarseness 1 year	Mid- two-thirds right cord	Squamous carcinoma Dec. 10, 1929	Sept. 26, 1929 Loc. Nov. & Coc.	10 days	None	Hoarse whisper	2 years
23.	Male, 63 yrs. Manager	Hoarseness 4 months	Anterior two-thirds right cord	Early squam. epitheloma Dec. 10, 1929	Dec. 17, 1929 Loc. Nov. & Coc.	10 days	None	Whisper Glottis normal	1 year and 9 months
24.	Male, 68 yrs. Gen'l Mgr.	Hoarseness 3 months	Anterior end left cord	Squamous cell carcinoma May 29, 1930	June 2, 1930 Loc. Nov. & Coc.	3 weeks granuloma	None	Good whispered voice	1 year and 3 months
25.	Male, 65 yrs. Ins. Agent	Hoarseness 1 year	Left cord	Squamous epitheloma May 3, 1929	Nov. 23, 1930 Loc. novocaine	None	Recurrence Partial laryngectomy Laryngectomy	No metastasis Larynx removed	1 year
26.	Fem., 26 yrs. Teacher	Hoarseness 2 years	Mid-third left cord	Squamous carcinoma Dec. 30, 1930	Jan. 17, 1931 Loc. Nov. & Coc.	2 weeks granuloma	None	Good whispered voice	9 months
27.	Male, 60 yrs. Salesman	Hoarseness 1 year	Anterior end right cord	Squamous epitheloma Feb. 17, 1931	March 9, 1931 Loc. Nov. & Coc.	3 weeks	None	Good voice Normal airway	6 months
28.	Male, 48 yrs. Cotton Buyer	Hoarseness 9 months	Anterior end left cord	Papilloma beg. cancer June 24, 1931	June 27, 1931 Loc. Nov. & Coc.	2 weeks	None	Good voice Normal airway	2 months
29.	Male, 65 yrs. None	Hoarseness 4 months	Anterior 3rd right cord	Cancer June 18, 1931	July 1, 1931 Loc. Nov. & Coc.	3 weeks	None	Good voice Normal airway	2 months
30.	Male, 72 yrs. Text. Carder	Hoarseness 2 months	Anterior end left cord	Squamous cell epitheloma Aug. 25, 1931	Sept. 3, 1931 Loc. Nov. & Coc.	2 weeks	None	Good voice Normal airway	Recent

blade of a turbinotome is inserted through the incision in the cricothyroid membrane and is carried upward inside the larynx until it is in contact with the base of the epiglottis. The outside blade being level with the thyroid notch. The thyroid cartilage is then clipped through, the wings retracted, the growth on the cord exposed and carefully examined. Then an incision extending through the mucous membrane completely surrounding the growth and about a half centimeter beyond the furthest extension of the growth is made. A careful dissection is then made, elevating the internal perichondrium of the thyroid wing on the affected side from the cartilage. A curved scissors is then used to cut through the mass of tissue from the incision in the mucous membrane, including the internal perichondrium, that has been dissected free. Hemorrhage is controlled by ligation and by suture ligation and pressure.

In the anterior commissure growth, the technic of Jackson (see Fig. 7) is followed. The cartilage is sawed through, using a Clerf saw. Great care is taken not to penetrate the internal perichondrium. To avoid this a subperichondrial dissection may be made by dissecting downward from the thyroid notch and upward from the lower border of the thyroid cartilage until the internal perichondrium is free from the anterior angle of the thyroid. A Gigli saw may be passed between the internal perichondrium and the cartilage, and the cartilage sawed through from inside, if desired. The thyroid wings are retracted (See Fig.) and the internal perichondrium separated to a point well beyond the margin of the growth, and the cancer bearing growth area with a wide margin of normal tissue is excised. Hemorrhage is controlled, and in cases where considerable of the mucous membrane of the epiglottis has been excised it is advisable to place a deep suture from the incision on either side just above the thyroid notch, passing beneath the base of the epiglottis. When this suture is tied it holds the base of the epiglottis forward and prevents it dropping back toward the posterior commissure and helps to maintain a good lumen through the glottis. Hemorrhage inside the larynx must be very carefully controlled before the larynx is allowed to close. This is usually not a difficult matter to accomplish, but should be very carefully done. The external wound

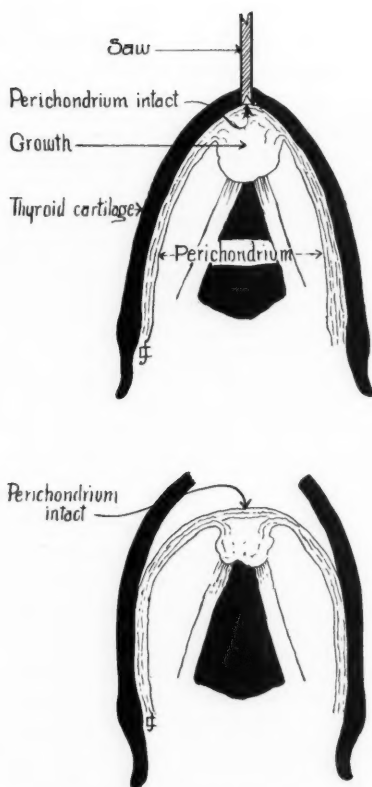


Fig. 7. Jackson's method of subperichondrial dissection in anterior commissure growths. Procedure in cases in which the cancerous growth is in the anterior commissure. The shears are not used for fear of cutting the growth. The thyroid cartilage is sawed through without injury to the inner perichondrium which is then dissected backward safely beyond the posterior limits of the growth.

is closed by interrupted silkworm sutures which include the skin and subcutaneous tissue and muscle down to the cartilage, but not including the cartilage. A strip of iodoform gauze is placed at the lower angle of the wound, securing it with a suture so that it cannot get into the larynx through the opening in the cricothyroid membrane. A second strip of gauze is placed in the middle of the incision beneath the sutures over the edges of the cartilage. The upper drain is removed in twenty-four hours. The lower angle of the wound is kept open until the incision in the cricothyroid membrane closes. Abundant dressings are applied to the external wound and are changed frequently as required. The patient is placed in bed with a backrest. If there is difficulty in swallowing, a small rubber catheter is passed through the anterior nares into the upper esophagus. Abundant fluids are given. Elderly patients are gotten out of bed within the first twenty-four hours. The sutures are removed, usually within four or five days. If infection develops in the wound, all sutures are removed. On appearance of infection, the wound is opened and is allowed to heal by granulation. If this occurs the convalescence is usually lengthened from ten days to three weeks. Otherwise the end result seems to be the same.

Complications.—Postoperative hemorrhage is a possible complication, and provision is made by this technic for the insertion of the tracheotomy tube through an incision of the tracheal rings which have already been exposed. The larynx may then be packed above the tracheotomy tube. The patient is encouraged to cough, and it is of very great assistance to the patient for him to place his hand over the outside of the dressing and hold the wings of the thyroid cartilage together when he attempts to cough. Morphin and all cough sedatives are very carefully avoided. Special precaution must be taken in regard to this, for all resident physicians are trained to give morphin postoperatively, and it must not be given to patients who have had external surgery of the larynx.

In a considerable percentage of the cases granuloma will form in the anterior commissure after the wound has closed externally. Occasionally a small piece of cartilage will necrose and

separate. This cartilage can be removed externally or by direct laryngoscopy. The granulomatous masses may require direct laryngoscopic removal several times during the first three or four months. It requires only a few minutes and is done with local anesthesia. The removed tissue is, of course, submitted to histologic study to be sure that we are not dealing with a recurrence. Tracheotomy is not done unless it is required to pack the larynx on account of hemorrhage or some undue reaction in the larynx.

The Technic of Sir St. Clair Thomson.—The essential difference in Thomson technic is that the thyroid wing on the affected side is dissected free of perichondrium, both internally and externally, and the freed portion of cartilage excised. This gives a much better exposure of the inside of the larynx and furnishes an excellent approach for partial laryngectomy if it is desired to carry out this procedure. In a number of cases not enumerated in this series a partial laryngectomy has been done by this method, and in some instances radium applications made inside the larynx, allowing the larynx to remain partially open, with excellent results.

CONCLUSIONS.

1. Early intrinsic cancer of the larynx will be recognized when the profession realizes fully the possible significance of chronic hoarseness. Biopsy by means of direct laryngoscopy should be the final step in the diagnostic study.
2. Early "anterior intrinsic cancer" of the larynx is amenable to cure by laryngofissure, which will save the patient's larynx as well as his life, with no operative mortality. The anterior intrinsic group includes a large percentage of all cases of cancer of the larynx.
3. Laryngectomy will cure practically all recurrences after laryngofissure if the cases are properly followed.
4. In twenty-five cases of the series reported, over one year has elapsed since laryngofissure. There have been two recurrences in two patients, giving a percentage of lasting cure of 92 per cent to September, 1931. One case of recurrence is well, without metastasis, following laryngectomy.

5. Partial laryngectomy with postoperative radiation has given good results where there was a contra-indication to laryngectomy in several cases not in this series.

6. Laryngectomy will cure a large percentage of posterior intrinsic cancer if the diagnosis is made while the lesion is still early.

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IV.

THE PHYSIOLOGY OF DRAINAGE OF NASAL MUCUS: II. CILIA AND MUCIN IN THE MECHANICAL DEFENSE OF THE NASAL MUCOSA: A MOTION PICTURE DEMONSTRATION.*

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This demonstration is a by-product of a systematic study of various phases of the common cold, begun about six years ago.

Nowhere in the body is there such a delicate structure, so greatly exposed to the environment, as the nasal mucosa. It meets the force of the inspired air sixteen or eighteen times a minute. This air varies tremendously in temperature and humidity, and carries with it dust, a great variety of bacteria and all manner of gases and fumes. It would seem that some strong, leathery, inert membrane would be necessary to withstand the rigors of inspiration. Instead, there is a highly specialized, frail epithelial membrane, covered with extremely fine and delicate cilia, the length of which is less than the diameter of a red blood cell.

A very remarkable mechanical defense has been provided to protect this mucosa from destruction. It consists mainly in the covering composed of mucinous secretion supplied by the many glands, and the rapid and effective ciliary motion that moves the mucin.

Rhinologists have not studied ciliary action with the zeal that biologists have displayed. In an academic way rhinologists know about ciliary activity, but in treatment they do not seem to take it very seriously. The purpose of this demonstration is to emphasize the force and importance of this phenomenon.

Cilia are rapid, powerful, effective and work independently of gravity. They move comparatively tremendous loads, and do so with dispatch. The action is automatic, like that of the heart

*Given before the American Academy of Ophthalmology and Otolaryngology, September 17, 1931. Work done in Section on Otolaryngology and Rhinology, The Mayo Clinic.

muscle,¹ and is not destroyed by fragmentation of the cell from which the cilia arise. A single cilium continues to beat if only a small bit of cytoplasm is attached. The cilia on each cell are beautifully coordinated in action. So are fields of cilia involving millions of cells.

This remarkable coordination, forming waves that pass rapidly over the surfaces, is one of the most striking characteristics of ciliary action. The power is very considerable. It has been found that the action increases with pressure up to a limit. In frogs' epithelium the optimal pressure is said to be 12 gm. to the square centimeter. Beyond this, activity decreases until it finally ceases at a pressure of 20 gm. to the square centimeter.¹ In the human being, the optimal pressure is said to be about 5 gm. to the square centimeter. The weight of as much mucin as would fill an antrum would not stop the ciliary activity on the floor of the cavity, but instead would serve to stimulate it. The rapidity of the beat is rather remarkable. It is generally about 250 cycles a minute in the nose of a man, but in some of the lower forms may go as high as 800 each minute. The mechanism of control is still under dispute. There is considerable evidence that cilia are under the control of both the autonomic and sympathetic systems.⁵ Parker did not accept this view, and more work doubtless will be necessary before the question is settled.

The protective function of the mucinous secretion is well known. It keeps the air from coming directly in contact with the surface cells. It entangles and encloses foreign particles and bacteria, thus preventing them from invading the cells. As the secretion is moved away toward the stomach, it carries any and all particulate matter with it.

The rate and manner in which the secretion moves is not so well known nor so fully appreciated. It forms a film that covers all the surfaces within the nose, sinuses, pharynx and esophagus, as a single, intact membrane. This film is in continuous motion throughout its extent, from the vestibule of the nose to the stomach. It moves over a fairly definite drainage system, characterized by great variations of speed. In the anterior third of the nose the speed is very low, and in the posterior two-thirds, com-

paratively great. On this basis the nasal surfaces may be divided into inactive anterior and active posterior regions.

In the latter region the ciliary activity is comparatively high, and the mucinous secretion flows along 4 to 6 mm. a minute. It usually requires less than ten minutes for drops of ink, placed on the surface, to traverse the distance of the posterior two-thirds of the nose to the nasopharynx. This means that this portion of the mucous membrane has an entirely new mucinous covering every ten or fifteen minutes. The rate of flow in the anterior third or quarter of the nose is not so great. Here, where the first impact of inflowing air is met, ciliary activity seems to be slight or absent, and the rate of flow is only a few millimeters an hour. Presumably, cilia are lacking and the drainage takes place by means of traction on the mucin contained in the secretion. That is, the film of secretion is drawn backward by the power of the active cilia in the posterior two-thirds of the nose. As it is moved backward it is drawn into the inferior and middle meatuses, where the mucous membrane is best protected and the ciliary action activity presumably is greatest. Hence, the anterior portion of the lateral wall, where dust and bacteria collect, drains through these two meatuses very largely. The posterior two-thirds drains backward toward the nasopharynx more directly. The mucus may enter the nasopharynx at the posterior border of the septum, the rostrum of the sphenoid, the soft palate or the lateral wall; wherever it enters, it is moved away to the stomach by the next act of swallowing.²

The drainage system of the frontal sinuses of twenty dogs was studied; most of the animals were operated on several times and inspected under various conditions. Hydrokollog was used as an indicator to show the rate and direction of the drainage. Small drops of this material were dotted here and there on the walls of the sinus and their actions observed.

It was surprising to see that the hydrokollog did not travel toward the ostium by the most direct routes. Instead it spiraled in a lateral direction. In general, the spirals began on the middle third of the mesial wall, passed posteriorly to the posterior wall, then laterally and slightly upward to the lateral wall, thence anteriorly and upward in an arc that eventually terminated at the

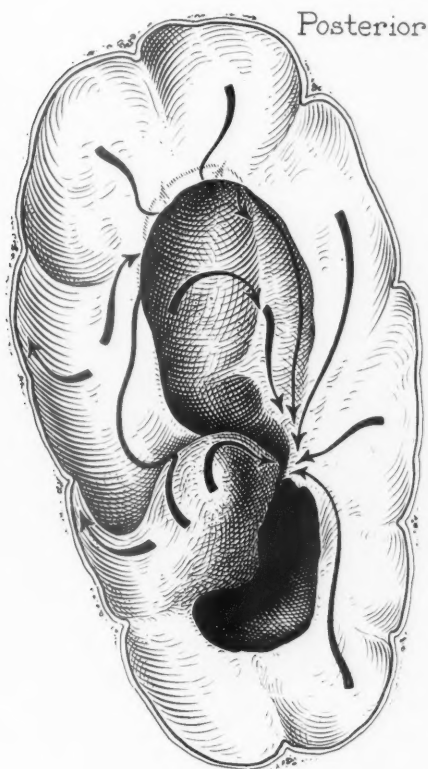


Fig. 1. The spiral direction of the drainage paths in the frontal sinus of the dog. From some areas the primary direction of drainage is away from the ostium.

ostium (Fig. 1). From the deepest recesses the direction was more directly toward the ostium and often almost against gravity. Lucas found a similar spiral type of drainage in the antrums of freshly killed monkeys.

There did not seem to be any inactive areas in this sinus that compared with those found in the anterior third of the nose of man, but the rate varied a great deal in different areas. There

were certain lines, or bands, along which the flow was faster than elsewhere. Areas lying close to these bands often drained into them by tributaries, as it were. Traction on the mucin by the more active cilia may account for this. The smooth spiral, described by a line of drainage passing in one revolution directly into the ostium from a distant point in the sinus, would seem to indicate a nearly impossible feat of coördination. To illustrate hypothetically, if the length of the cilia is increased to that of wheat stalks in a field and the area of the sinus is represented by a wheat field of corresponding size, the field would be approximately seven miles long and five miles wide, and the ostium a small gorge at one end. Suppose that the wheat moved with the motion of cilia and passed articles along over their tops in the manner of cilia. To simulate the action in the sinus, an object starting near one corner of this immense field would pass around the entire field in a single tremendous circle several miles in diameter, and arrive accurately, and pass out at an exit comparatively small. This would represent an amazing accuracy of coördination and direction of ciliary movement. As a matter of fact, there is a gradient in the speed of flow which reduces the necessity for accuracy. As the spiral approaches the ostium, the rate of flow becomes progressively greater. The flow along a given continuous drainage arc may be several times as fast as the ostium as in a distant part of the cavity. If this increased speed is due to greater ciliary activity, as presumably it is, then the mucinous layer would tend to move toward this area of greatest activity at the ostium by means of traction, even though the direction of the ciliary beat were somewhat inaccurate.*

The rate of flow varies widely. The greatest rate measured in a normal sinus was 18 mm. each minute. In a sinus the ostium of which had been completely closed at a previous operation several weeks earlier, the high rate of 24 mm. for each minute was found. Some of the variations found might be accounted for by the ether used as anesthetic. When the tracheal tube, through which the ether was administered, failed to fit the trachea closely, leakage of the anesthetic through the open sinuses sometimes

*A more detailed account of the drainage of the dog's frontal sinus, including measurement of rate of flow, will be published soon.

occurred. Ether will abolish ciliary activity when sufficiently concentrated.

The direction of flow was found to be independent of gravity. In fact, the path of greatest speed was often found to be in the line between the deepest recess and the ostium. The direction here is almost vertical. As a matter of fact, the major portion of our own respiratory tract drains through the trachea directly upward.

The mucous membrane of a dog's frontal sinus, when normal, is so extremely thin that it looks as frail as a soap bubble. When stripped out completely from a cavity which is about as large as the antrum of man, it occupies but a minim or two of volume. The secretion covering it is so thin that it is practically imperceptible. Yet the effectiveness of this delicate mechanism in emptying the cavity is amazing. Since the object of this demonstration is to give an impression of this effectiveness, I shall cite the results of a few experiments.

A series of dogs was prepared for injection of fluids through hypodermic needles placed in the frontal sinuses. The bone covering the sinuses was removed at operation, preserving the lining membrane as much as possible. The skin was then closed over the mucous membrane and the wound allowed to heal. When healed, injections could be made readily, directly into the cavity, through fairly large needles.

A series of injections was made for the purpose of infecting the sinuses. The following injections failed: Cultures of laboratory strains of streptococci, cultures of streptococci derived from victims of respiratory infections, cultures of pneumococci from respiratory infections, mixtures of streptococci, pneumococci and several other virulent organisms, suspensions in saline solution, centrifuged concentrates as thick as cream, pus from various infections, fresh nasal secretion from a patient in the second day of a cold, and finally, sand mixed with some of the foregoing organisms. When some of these sinuses were opened after twenty-four hours, they were uniformly as white and the membranes grossly as thin and transparent as before. One sinus was opened under anesthetic and observed directly as it rid itself of a thick mucus. A comparatively

large mass of very heavy mucilaginous mucus was dropped into the cavity. In fifty minutes the last trace of it had disappeared.

SUMMARY.

Ciliary action and a protective, moving film of mucin over the surface are the chief mechanical factors involved in the drainage and defense of the nasal mucosa. The drainage, as activated by the cilia, is rapid, powerful and effective. Gravity plays a very minor part. Traction, on the other hand, exerted on the film of mucin as on a net is a very important factor in drainage. A complete exchange of the film of secretion over the surface takes place every ten or fifteen minutes in the more active regions in the nose, and about once an hour in the inactive regions. The drainage is directed backward with a strong tendency to flow toward the best protected areas; namely, the middle and inferior meatuses. The inactive anterior third of the nose drains largely through these meatuses by means of traction. The sinuses (as represented by the frontal sinus of the dog) have definite and extremely efficient drainage that is spiral in direction.

With these points in mind, the necessity of changing the viewpoint of our treatment of nasal conditions from an anatomic to a physiologic one becomes apparent.³

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V.

THE SURGICAL TREATMENT OF CERTAIN UNUSUAL
CONDITIONS OF THE FRONTAL SINUS.*

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Certain very interesting problems arise in the satisfactory surgical treatment of unusual pathologic conditions of the frontal sinus. Good results depend on the employment of the correct type of operation for the particular case involved. Up to the present, no one surgical procedure has been advocated which fulfills all requirements. An operation that might be considered radical for one type of lesion might be considered conservative for another.

It may be said, in general, that when the external table of the frontal sinus is diseased, operation other than to remove the outer wall and obliterate the cavity, cannot be expected to effect a cure. Tumors filling the interior of the sinus must be approached in the same way. If the external table is not diseased, although the floor of the sinus may have perforated or have been destroyed, the external table does not necessarily need to be removed. In deciding on the type of operative interference indicated, several significant factors other than the pathologic process need to be considered.

1. *The Relative Danger to Life from the Operation Compared to the Danger of the Disease.*—If a case is encountered in which infection of the frontal sinus involved the bone of the anterior table, or if symptoms of early extension of the infection to the meninges were present, one would not hesitate to advise radical operation, even though the result caused some cosmetic disfigurement. One would hesitate to advise a disfiguring operation, on the other hand, if such symptoms and signs were absent. If, for instance, roentgenograms of the head revealed the presence of a

*Read before the Midwestern Section of the American Laryngological, Rhinological and Otological Society, Kansas City, Missouri, January 21, 1932.

Section on Otolaryngology and Rhinology, The Mayo Clinic.

small osteoma in the frontal sinus and there were no symptoms, the danger of an operation might be greater than the risk of observing the course of the growth. Certain osteomas do not increase in size or cause symptoms.

2. *Social Status of the Patient.*—Because the face and forehead are exposed to view, patients are naturally somewhat sensitive about the effect that deformity may have on their friends, new acquaintances and on their own mental reaction. This is particularly true of young men and women just about to start on their careers, as the deformity may become a severe handicap socially and financially. Under such circumstances, much consideration must be given to the prevention of undue deformity. Fortunately, even the so-called obliterating operation can be employed without much disfigurement. I should like to emphasize that after all factors have been taken into consideration, it should be clearly understood that if the operation revealed conditions that could not be safely cared for as originally planned, then the surgeon must be allowed to use his own judgment without reservation.

The material to be considered here consists of cases presenting quite similar symptoms and physical findings for which different types of operations were used. For obvious reasons, an effort will not be made to detail exhaustively pathologic, anatomic or clinical factors. The following two cases of pyoceles of the frontal sinus present similar symptoms, and data concerning them have proved interesting and instructive.

A young man, aged twenty-three years, complained of swelling in the region of the left upper inner canthus, of five months' duration. The left eye was displaced downward and outward. However, diplopia was not present. The movements of the eye were limited upward; pathologic changes of the fundus were not noted and vision was not affected. Definite pathologic changes were noted of the lateral wall intranasally. Pressure on the mass in the orbit caused thick pus to enter the nostril. Palpation revealed that there was apparently no floor to the frontal sinus. A diagnosis was made of suppurative frontal sinusitis with pyocoele. A two-stage intranasal and external operation was advised (Fig. 1). The intranasal operation consisted of re-



Fig. 1. Floor of frontal sinus bulging into the orbit (Case 1).

removal of the middle turbinate and forcibly making a large opening with a gouge through the dense bone bulging into the region of the agger nasi. The ordinary instruments for entering this region were ineffective. The opening was greatly enlarged. It was readily recognized that the pyocoele had by pressure caused absorption of the thin floor of the frontal sinus and the paper plate of the ethmoid sinus. Complete relief of the symptoms followed and the external operation was not necessary.

A man, aged thirty-three years, complained of swelling of the left upper eyelid and bulging of the eye of six months' duration. Disease of the sinuses had been intermittent for six years. Pressure on the swelling did not change its appearance and pus did not escape into the nose. A dull headache was becoming increas-



Fig. 2. Condition of the left frontal sinus and fronto-ethmoid cells (Case 2).



Fig. 3. Osteoma in the left frontal sinus (Case 3).

ingly troublesome. The right eye appeared to be deeply set in comparison to the prominent left eye. There was a large swelling in the upper lid which fluctuated but did not evacuate on pressure. The floor of the frontal sinus could not be felt. The exophthalmometer reading of the right eye was 12 and of the left eye, 13. Vision in the right eye was 6/10 and in the left 6/15. Definite fullness of the left agger nasi and region of the bulla was noted. The frontal sinus could not be probed. Pus did not appear in the nostril after pressure on the mass. Marked cloudiness could be seen in the roentgenogram of the maxillary and frontal sinuses (Fig. 2). A two-stage intranasal and external operation was advised. At operation the region of the bulla was easily broken into because the bone was very thin. A large amount of thick



Fig. 4. Effect of operation (Case 3).

foul-smelling pus escaped immediately. Pressure on the mass in the orbit increased the amount of discharge. On collapse of the mass, a defect in the floor of the frontal sinus could be felt. The opening into the nose was greatly enlarged. Following the operation, the swelling of the lid and displacement of the eyeball were relieved. Foul-smelling pus continued to drain from the frontal sinus for about three weeks. An external operation was performed and it was found that the cavity of the pyocoele was lined with thick inflammatory tissue. This was carefully removed. The outer portion of the cavity caused by the pyocoele was at a lower level than the nasal outlet, due to the extension of the fronto-ethmoid cell over the globe. The Lynch type of operation was used. The result appeared to be satisfactory for about six

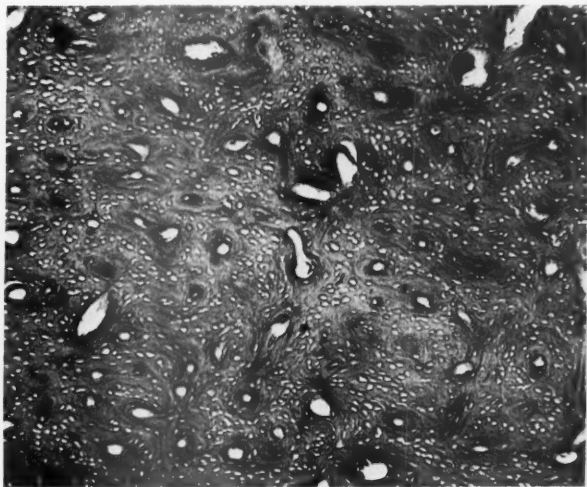


Fig. 5. Pathologic characteristics of osteoma (Case 3).

months, then the large intranasal opening closed and symptoms recurred. The obliterating Killian operation was decided on, and the result was very satisfactory, as the disease was controlled with practically no resulting disfigurement. The patient has been observed at intervals and the condition remains good.

Just why the first patient recovered following the intranasal operation and the second patient did not cannot be explained satisfactorily. Essentially the same type of operation was used, but the obliterating Killian operation was necessary to effect cure of the second patient.

3. *Changes in Bone Within the Frontal Sinus.*—It may be difficult to arrive at a definite opinion concerning changes occurring in bone within the frontal sinus unless pathologic specimens are studied. The following two cases present similar symptoms and signs but different pathologic characteristics.

A woman, aged forty-two years, was seen in consultation because a roentgenogram of the frontal sinus was interpreted as showing a suppurative condition requiring operation. The facts

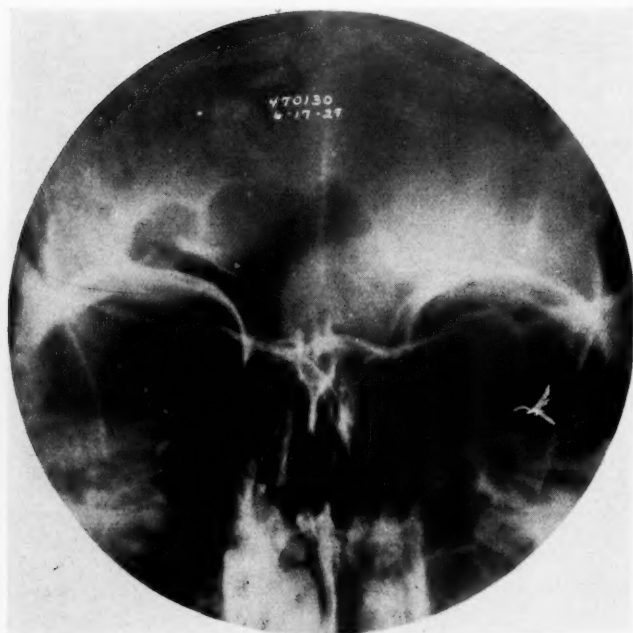


Fig. 6. Changes in bone in frontal sinus before operation (Case 4).

were that as a result of acute infection of the upper part of the respiratory tract, the patient complained of headache (Figs. 3 and 4). The roentgenogram actually revealed an osteoma of the frontal sinus. Recovery from the respiratory infection was followed by relief of the headache. Observation of the patient over a period of two years revealed that the osteoma was increasing in size with increase in symptoms, especially mental anxiety, and headache. Removal of the osteoma relieved the patient (Fig. 5).

A physician, aged thirty-three years, had discovered by roentgenographic examination that some peculiar changes had taken place in his right frontal sinus. He had noticed that with infection of the upper part of the respiratory tract, headache over the frontal sinus had become increasingly troublesome. The head-



Fig. 7. Frontal sinus after operation (Case 4).

ache was not actually relieved when the respiratory infection had subsided. Closer questioning revealed that the right side of the forehead had been injured in an automobile accident a few years before. The roentgenogram (Figs. 6 and 7) showed a dense shadow in the frontal sinus not entirely filling the sinus to the outlines. The shadow resembled osteoma closely. The sinus extended well over and above the median line, and beyond the mass it was cloudy as compared with the small frontal sinus on the opposite side. The intranasal operation was performed without incident. The external operation was performed after the method of Lynch to avoid deformity. It was found that the mass within the sinus was highly differentiated bone, and under the conditions it was difficult to remove. Above the mass, the secretions from



Fig. 8. Pathologic characteristics of highly differentiated bone (Case 4).

the membrane had accumulated and produced a mucocele. The mass was removed with satisfactory results. A pathologic diagnosis was made of eburnation (Fig. 8). The probabilities are that the eburnation was the result of scarring of bone from the injury plus the osteoplastic effect of the presence of low-grade infection. The same effect is observed in chronic mastoiditis, but in my experience it is rare in the paranasal sinuses.

4. *Foreign Substances Within the Frontal Sinus.*—Bismuth paste was advocated several years ago for the cure of certain suppurative lesions. I have encountered this paste only once in a suppurative frontal sinus.

A woman, aged fifty-eight years, presented herself because of a draining fistula from the left frontal sinus. An external frontal operation had been performed six months previously because of



Fig. 9. Presence of foreign body which proved to be bismuth paste (Case 5).

swelling over the forehead, and headache. Examination disclosed a fistula into the left frontal sinus, the probing of which caused flakes of bismuth paste to escape. The nasofrontal duct had apparently not been enlarged, as a very small probe could not be passed. The two-stage operation was advised. The intranasal operation was accomplished without difficulty and the nasofrontal duct was greatly enlarged. The bismuth paste could not be removed (Fig. 9). The external Killian operation was performed later and the result was excellent.

A man, aged fifty-three years, complained of severe frontal headache. He had had purulent discharge from the nose for about twenty years. With each cold in the head, the headache

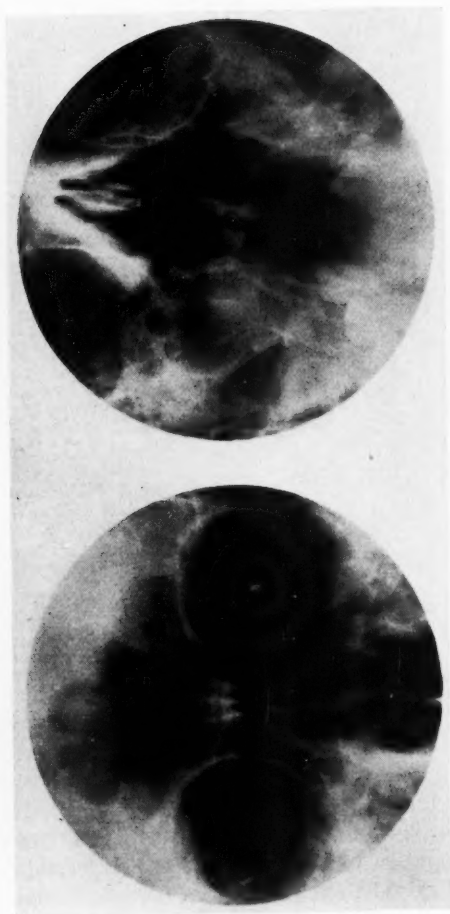


Fig. 10. Caseous mass is not shown in the roentgenogram (Case 5).

became increasingly more severe until he was unable to carry on his work. Examination revealed evidence of suppurative pansinusitis (Fig. 10). An extensive intranasal operation relieved the symptoms. Eight years later the patient returned, stating that for three years there had been some discharge but little annoyance. Three weeks previously the eye became swollen and the headache recurred. Examination at this time disclosed a large fluctuant swelling of the upper lid. Intranasally, the nasofrontal duct, which had been greatly enlarged at the operation eight years previously, was found apparently closed. A Lynch type of operation was performed. Within the sinus was found a large, foul-smelling, cheesy mass resembling cholesteatoma. The cavity was carefully cleaned and the external wound closed. The condition seemed to be well controlled and the patient was dismissed in four weeks. He returned in six months with a draining external fistula. A Killian operation was performed and the patient has been well for three years.

I have observed caseous, foul-smelling masses in the antrum but I have not observed a layered mass. The question arises whether the mass in the frontal sinus was caseated pus or whether it was cholesteatoma. As the result of the closure of the nasofrontal duct, it is conceivable that nests of epithelial cells could have produced the mass in the same way that masses are formed in the mastoid. Bismuth paste, as a curative agent, apparently is not effective in a cavity in which the walls do not collapse.

5. *The Frontal Sinus Draining Directly Into the Maxillary Sinus.*—Anatomic specimens occasionally reveal an accessory duct from the frontal sinus that communicates directly with the maxillary sinus. Only once in my experience have I encountered a clinical case in which the maxillary sinus was acting as a reservoir for mucopurulent secretion from the frontal sinus.

A man, aged thirty-eight years, complained of pain over the right frontal and maxillary sinuses and purulent discharge in the nose. The symptoms had followed infection of the upper part of the respiratory tract about three years previously. Several intranasal operations had been performed without relief. Since the operations he had noticed occasionally a foul odor. The right

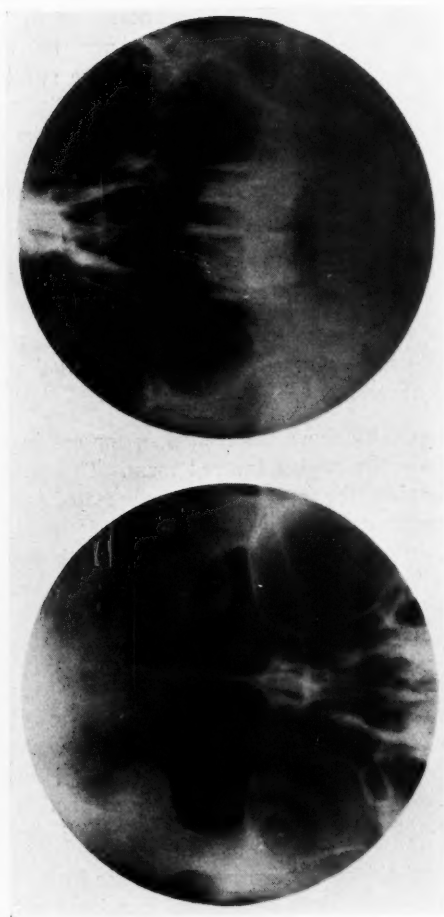


Fig. 11. Maxillary sinus acted as a reservoir for discharge from the frontal sinus (Case 7).

antrum had been opened and contained much mucopurulent discharge. The middle turbinate had been partially removed and the anterior ethmoid cells opened. What was thought to be the nasofrontal duct could be probed. The region was covered with mucopurulent secretion. Roentgenograms showed the frontal and maxillary sinuses to be cloudy compared to the left side (Fig. 11). It was decided to investigate the sinuses and make the observations necessary to arrive at some definite opinion regarding treatment. The observations led me to believe that the maxillary sinus caused the symptoms. An intranasal operation did not relieve the symptoms, so an alveolar operation (Caldwell-Luc) was performed. This revealed a pocket containing pus, situated laterally below the floor of the orbit. It was difficult to remove the thickened membrane. In the postoperative care of the patient it was noted that the amount of discharge did not decrease. The antrum would be found well filled an hour or more after it had been emptied. The possibility of the antrum acting as a reservoir was thought of. The Lynch type of operation of the frontal sinus was then performed. The frontal sinus and fronto-ethmoid cells were found filled with thick, mucopurulent secretion. A fronto-ethmoid cell opened directly into the maxillary sinus and there was no opening into the nose. What appeared to be the nasofrontal duct from below was in reality an ethmoid cell. Recovery was rapid and complete. The patient has been observed twice in the five years that have elapsed and he has remained well.

SUMMARY.

Seven case histories of patients presenting interesting problems of the surgical treatment of unusual diseases of the frontal sinus are presented. Even though the clinical and physical data might be similar, it was found that they did not respond to the same type of surgical interference. It is evident that the time honored surgical axiom that the operation must be adapted to the pathologic condition rather than the pathologic condition to the operation holds true in this group of cases.

VI.

MALIGNANT TUMORS OF THE NASOPHARYNX WITH INVOLVEMENT OF THE NERVOUS SYSTEM.*

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Malignant tumors of the nasopharynx, arising from Rosenmüller's fossa or from the vault, produce a great variety of symptoms and signs, which result from extension of the tumor into the cavity of the nasopharynx and into the adjacent anatomic structures. For all practical purposes, these symptoms and signs may be divided into the nasopharyngeal and the extranasopharyngeal. In view of the fact that this presentation concerns the involvement of the nervous system, the symptoms and signs exhibited must arise from the extranasopharyngeal extension of these tumors. Symptoms referable to the nasopharynx are often absent; therefore, diagnosis is not always easy, and the primary lesion may not be particularly looked for in the nasopharynx unless the involvement of the cranial nerves suggests this possibility.

In reviewing the literature on this subject, it is remarkable to note that with the following exceptions comparatively few cases have been reported in which the diagnosis was based primarily upon involvement of the nervous structures. The most outstanding and extensive observations on this subject are those of New,¹²⁸ who in his last publication, in 1925, reported 119 cases and to date has observed approximately 200⁴ cases. He has called attention to the relation of nasopharyngeal malignancy to other diagnoses, has emphasized the frequency of early extranasopharyngeal symptoms and has pointed out the need of a better understanding of this syndrome.

*Presented before the fifty-third annual meeting of the American Laryngological Association, Atlantic City, New Jersey, June 15-17, 1931.

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In 1922, Woltman⁵ published a very comprehensive report on a group of 25 cases with involvement of the nervous system which were observed with New and which were selected from the latter's group of 79 cases. Woltman also collected reports of 44 similar cases from the literature. A review of the papers of New and that of Woltman gives a very comprehensive understanding of the various clinical pictures encountered with this type of lesion.

The material for this presentation consists of a group of nine cases with involvement of the nervous system, selected from a series of seventeen cases. From a previous report⁶ of twelve cases of malignant tumors of the nasopharynx, four cases have been reconsidered from the standpoint of involvement of the nervous system. The clinical picture may be clearly understood by a consideration of the usual location of the primary tumor, the various structures involved by its extension and the various symptoms and signs produced thereby.

The tumor is most frequently found in the fossa of Rosenmüller and in the vault. By extension laterally the adjacent structures, such as the eustachian tube and the cranial nerves, may become extensively involved. The bony structures in this region, being made up of thin layers of bone, are easily eroded. The foramina in the base of the skull and the sphenoid fissure may be invaded. The sphenoid and ethmoid sinuses may also be affected. When the dura is reached it may be elevated but it is rarely penetrated. Even the pituitary region may be invaded. Cervical metastases are common and sometimes distant metastases are noted.

As previously remarked, symptoms referable to the nasopharynx may be absent. In six of the nine cases these symptoms were absent. In sixteen of Woltman's twenty-five cases they were also absent. Pain the eye, side of the face and ear were the most common complaints. Deafness and tinnitus from involvement of the eustachian tube were present in six cases. Symptoms referable to the eye, such as blindness, ptosis or diplopia, were present in eight of the nine cases. Fifth nerve involvement was apparent in four cases and seventh nerve paralysis in two; the jugular foramen syndrome was complete in three cases and partial in one. (See Table 1.)

TABLE I.

CRANIAL NERVE INVOLVEMENT IN NINE CASES OF MALIGNANT
TUMORS OF THE NASOPHARYNX.

Case	Age	Sex	Type of Tumor	First	Second	Third	Fourth	Fifth	Sixth	Seventh	Eighth	Ninth	Tenth	Eleventh	Twelfth
1. R. W.	39	M	Ca.								*	x	x	x	x
2. C. C. M.	54	M	Lym.						x		*	x	x	x	x
3. J. R. G.	53	M	Ca.					x	x		*				
4. A. B.	44	M	Ca.						xx						x
5. H. R.	24	M	Ca.		x		x			x	*				
6. F. S.	42	M	Ca.		x	x	x	x	x						
7. A. B.	50	F	Sq. Ca.			x					*				
8. N. L.	17	M	Sq. Ca.			x		x							
9. S. A.	42	F	Sq. Ca.		x	x	x	x	x	x	*	x	x	x	x

The sixth nerve is the most commonly affected (five cases) and is usually the first to show evidence of paralysis. Woltman reported sixth nerve paralysis in eighteen of twenty-five cases. The position of the sixth nerve in relation to the other nerves passing through the sphenoid fissure is variable, but, according to Sluder, it usually takes the lowest position. The sixth is also known to be very susceptible to pressure changes.

The extension of these growths into the sphenoid fissure and orbit give rise to a great array of symptoms and signs, such as pain in the eye, choked disc, blindness, optic atrophy, diplopia, ophthalmoplegia, exophthalmos, ptosis and constriction of the pupil.

Symptoms resulting from involvement of the fifth nerve are common and may simulate tumor of the gasserian ganglion. Both motor and sensory branches may be affected. In a report of four cases of tumor of the gasserian ganglion, Shelden⁷ called attention to the fact that the ganglion may sometimes be involved from tumors arising in the nasopharynx. The clinical manifestations are those of intracranial disease and may be indistinguishable from those caused by tumors arising within the cranial cavity and involving the ganglion.

The pain in the area of the fifth nerve varies in intensity and constancy. The pain is not identical with tic douloureux and is not induced by the same factors. There are changes in sensi-

bility to pain, touch and temperature. Paralysis of the motor branch of the fifth may be manifested by weakness of the muscles of the jaw. Ankylosis of the jaw may result from direct involvement of the pterygoid and masseter muscles.

The nasal ganglion syndrome with pain in and around the eye, forehead, temporal, mastoid and occipital regions and radiating to the neck, shoulders and arm is not uncommon.

The seventh nerve was involved in two cases. In one case it was associated with extensive invasion of the cervical glands and in the other case it was involved in association with the jugular foramen group of nerves. Direct involvement of the eighth nerve has not been reported. Although deafness and tinnitus are common, these symptoms can usually be attributed to involvement of the eustachian tube. In one case in which all the cranial nerves were involved on one side the deafness was very marked.

The ninth, tenth, eleventh and twelfth nerves are probably affected by glandular involvement but may be directly invaded by the growth. Palatal immobility may be caused by direct pressure from the tumor or by involvement of the levator palati muscle, as well as by nerve injury. In three cases the jugular foramen syndrome was complete and in one case the twelfth nerve alone was affected.

From the standpoint of the microscopic pathology of malignant tumors of the nasopharynx, several different types have been recorded, such as squamous cell epithelioma, adenosarcoma, lymphosarcoma, sarcoma, mixed cell sarcoma and carcinoma. Several types of carcinoma have been described, such as unclassified carcinoma, transitional cell carcinoma (Quick), lympho-epithelial carcinoma. In this group of cases, five were carcinomas, three were squamous cell epitheliomas and one lymphosarcoma. Squamous cell epitheliomas in the nasopharynx usually show a very high degree of malignancy, therefore very little tendency to differentiation. The carcinomas show a striking lack of differentiation. Pearl formation, spines and hornification are absent. The cells are small, having large nuclei and little cytoplasm. Mitotic figures are numerous. The intercellular substance is scanty and often there is a great deal of lymphocytic infiltration. The lympho-epithelial type of tumor, described by Ferreri,⁸

Jovin,⁹ Ewing¹⁰ and Marx-Münster¹¹ appears to be a type of carcinoma with marked lymphocytic infiltration. New classifies this type as a grade four squamous cell epithelioma (Broders). The radiosensitivity of these very malignant carcinomas has been discussed by several authors, particularly by Quick and Cutler.¹²

CONCLUSIONS.

The early diagnosis of malignant tumors of the nasopharynx is often difficult on account of the small size of the primary growth and the lack of nasopharyngeal symptoms. Extranasal symptoms are commonly present in the early stages of the disease. The early signs and symptoms are usually pain in the eye, forehead, side of the face and temporal region, toothache, earache, deafness, tinnitus, sense of fullness in the ear, diplopia, blindness, proptosis, paresthesia of the face, enlarged cervical glands, dysphagia, aphonia, hoarseness and distant metastases.

The cranial nerves are usually involved extracranially. Those passing through the sphenoid fissure are affected most commonly, particularly the sixth nerve. The fifth nerve is next most commonly involved.

The nasopharynx should always be most carefully examined in all patients with unexplained palsy or irritation of the cranial nerves in patients with enlargement of the cervical glands and in those with metastatic malignant disease in which an apparent primary lesion is not present.

Pathologically malignant tumors of the nasopharynx present a highly malignant type of lesion with a striking lack of cellular differentiation and offer difficulties of exact classification.

REPORT OF CASES.

Case 1.—A colored man, aged 39, was seen on July 25, 1925, complaining of hoarseness, difficulty in swallowing, pain and deafness in the left ear. These symptoms had been present for two months. He had been previously treated for cystitis and syphilis.

Examination of the larynx showed a complete paralysis of the left vocal cord, anesthesia of the left half of the larynx and partial paralysis of the soft palate, pharyngeal constrictors and

tongue on the left side. The nasopharynx showed a small grayish colored growth about 0.5 cm. in diameter in Rosenmüller's fossa on the left side. The left ear drum was normal, but a mild conduction type of deafness was present. In the upper deep cervical region on the left side behind the angle of the jaw an indefinite thickening but no definite gland was found.

Neurologic examination gave negative results except for paralysis of the cranial nerves as mentioned. The eyes were normal.

The patient was again examined two months later. A mass of hard, immovable glands was present in the upper deep cervical region on the left side. A small nodule was present in the skin over the triceps muscle of the left arm. Complete paralysis of the ninth, tenth, eleventh and twelfth cranial nerves was found. The tumor in the nasopharynx increased to about 1 cm. in diameter. It was grayish and nonulcerated. A piece of it was removed for diagnosis, and it proved to be carcinoma.

The nasopharynx was treated with 500 mg. hours of radium, and deep X-ray therapy was applied to the neck. This did not prevent the progress of the lesion. Within the following month the patient developed blindness in the left eye, with complete ophthalmoplegia and paralysis of the fifth, seventh, ninth, tenth, eleventh and twelfth cranial nerves. He died about two months later.

Comment.—Symptoms on the nose and pharynx were not present in this case. When the primary growth was discovered, it was small (less than 0.5 cm. in diameter), while the paralysis of the ninth and tenth cranial nerves was complete. This case emphasizes the importance of examination of the nasopharynx of persons with paralysis of the cranial nerves.

Case 2.—C. C. M., a white man, aged 54, was seen by a general surgeon on June 7, 1924, because of enlarged glands of the neck, which had been present for about a year. The tonsils had been removed previously without causing any change in the condition of the glands.

A tentative diagnosis of Hodgkin's disease was made. Symptoms in the nose, throat or ear were not present. A gland was removed for diagnosis and the pathologic report was as follows: Lymphoid proliferation, with thickening of the capsule and an

increase of connective tissue, with an occasional rather large cell, which is, no doubt, endothelial. The diagnosis was probable Hodgkin's disease. The glands were subjected to intense X-ray therapy and they gradually disappeared.

The patient was then free from symptoms for several months. On August 29, 1925, fourteen months after the gland had been removed for diagnosis, he was referred to me for the first time on account of earache, deafness and nasal discharge on the left side. These symptoms had been present for about four months. He also had pain in and around the left eye, which radiated to the temporal region. The pain in the ear was aggravated by movements of the jaw. He was unable to sleep on account of pain. The left side of the neck felt stiff and movements of the head were limited.

Examination of the nose showed purulent crusts in the posterior part of the left side. The entire left side of the nasopharynx was ulcerated and covered with a crust. There was a great deal of scar tissue along the lower border of the ulcer, which probably resulted from the X-ray therapy. The eustachian tube could not be identified. Glands were not palpable in the neck, but the upper deep cervical region on the left side was markedly thickened. The left ear drum was retracted and moderate conduction deafness was present. Tissue was taken from the ulceration but was unsatisfactory for examination. On the basis of the ulceration in the nasopharynx and the history of the enlarged glands, a tentative diagnosis of primary lymphosarcoma of the nasopharynx was made.

The patient returned six weeks later, complaining of more severe earache, pain in the eye and double vision. The examination of the eye showed paralysis of the sixth nerve. The ulceration in the nasopharynx had not changed. There was a slight paralysis of the pharyngeal constrictors and the soft palate. The nasopharynx was treated with radium (400 mg. hours). During the next two months the pain in the eye and ear was greatly relieved.

Three weeks later the patient returned complaining of pain in the left temporal and parietal regions, sensory disturbances in the second and third divisions of the fifth nerve and difficulty in swal-

lowing. The paresis of the soft palate and pharynx had become more marked, and partial paralysis of the left side of the tongue appeared. The larynx was normal.

One month later, pain in the temporal region and the left eye was severe. The fifth, sixth, ninth, tenth, eleventh and twelfth cranial nerves were now involved. X-ray and radium treatment did not stop the progress of the disease, and the patient died about two months later. The gland which had been removed from the neck was resectioned, and a diagnosis of lymphosarcoma was made.

Comment.—Primarily, the patient did not show symptoms in the ear, nose or throat, and sought treatment for enlarged glands of the neck. This case illustrates the importance of early examination of the nasopharynx in all patients with enlarged glands of the neck.

Case 3.—J. R. G., a white man, aged 53 years, was seen in consultation with Dr. Sachs on March 29, 1926. His illness had begun fourteen months previously with double vision, pain in the left eye and forehead, and toothache in the teeth of the left upper jaw. Several teeth had been extracted, and an operation had been performed on the nose, without relief. The pain in the eye was described as smarting or burning. The pain in the forehead was dull and constant. Five months before, he began to have pain in the back of the head and down the left side of the neck. He also had some slight dizziness and he said that his head felt heavy.

Three months before he entered the hospital, he began to have a dull earache and ringing in the left ear and a sensation of numbness along the zygoma. A lump in the left side of the neck had been noted for two or three months. Symptoms were not present in the nose or throat. Examination of the eye showed paralysis of the left external rectus. Slight general contraction of the visual fields was present; the fundus was normal.

Examination of the fifth nerve showed hypalgesia of the second and third divisions of the left side. The temperature sense was diminished.

On the left side of the neck, just behind the middle of the sternomastoid muscle, there was a hard, fixed gland about 6 cm.

in diameter. On the left lateral wall of the nasopharynx, in the fossa of Rosenmüller, a small growth or bulging of the wall, about 1 cm. in diameter, was present.

The neurologic examination gave negative results. The Wassermann reactions with the blood and spinal fluid were negative. A stereoscopic X-ray of the skull was negative.

A section of the tumor in the nasopharynx was removed for diagnosis and found to be carcinoma.

The tumor in the nasopharynx was treated with radium (400 mg. hours). Two months later, there was no evidence of it in the nasopharynx.

During the subsequent eighteen months this patient was kept under close observation. The gradual involvement of the orbit and cranial nerves was noted. About two months after he came under my observation, examination of the eye showed complete ophthalmoplegia, beginning optic atrophy and slight exophthalmos. The second and third divisions of the fifth nerve and the motor branch were completely paralyzed.

About a year later, a paralysis of the jugular foramen group of nerves began and was complete within a few months' time. The facial nerve was paralyzed.

At the time of his death, in September, 1927, all the cranial nerves with the exception of the eighth nerve were paralyzed on the left side.

Repeated treatment of the glands in the neck with X-ray was instituted but did not stop the progress of the lesion. The growth in the nasopharynx, however, never recurred.

Comment.—At the onset of the patient's illness, the only symptoms present were headache and toothache. An operation had been performed on the nose, and several teeth had been removed from the left upper jaw. When he came under my observation the presence of the nasopharyngeal tumor was suggested on the basis of the symptoms in the ear, paralysis of the sixth nerve, disturbance in the fifth nerve and a malignant gland in the neck. Symptoms in the nose or nasopharynx were not present.

Case 4.—A. B., a white man, aged 44, entered Barnes Hospital on July 15, 1927, complaining of severe headaches which had been

present for four months. He also complained of his tongue being "thick" and attacks of pain in the eye with radiation to the occipital regions. About six weeks previously double vision had appeared, and there was also a sense of fullness and impaired hearing in the left ear.

The patient appeared drowsy and at times incoherent. His speech was slightly impaired. He held his hand over one eye to relieve the double vision.

Physical examination: The patient was poorly nourished and somewhat emaciated. Bilateral paralysis of the sixth nerve was present. The right half of the tongue was partially paralyzed. The eyegrounds were normal. A small, hard gland was present in the upper left cervical region. Neurologic examination gave negative results. The examination of the ear, nose and throat showed a slight conduction deafness in the left ear. In the vault of the nasopharynx there was a small crust, beneath which a superficial erosion of the mucous membrane was noted. This was more marked on the left side than on the right side. In the upper part of Rosenmüller's fossa, on the left side, a small mass about the size of a pea was noted on the border of the erosion.

The nasopharynx was difficult to examine, as the patient co-operated poorly. A definite tumor was not present, and a diagnosis of malignant disease of the nasopharynx did not seem warranted.

A neurologic surgeon performed bilateral ventricle puncture and explored the region of the right temporal lobe. Nothing of significance was found.

It was decided to remove the gland in the left side of the neck for examination. This was done, and microscopic examination showed carcinoma. Then a biopsy specimen was taken from the area in the nasopharynx. This proved to be carcinoma.

The nasopharynx was treated with radium (400 mg. hours), and both sides of the neck were treated with deep X-ray irradiation. This patient returned home and has not yet returned for observation.

Comment.—The paralysis of the sixth nerve and of the twelfth nerve on the right side, together with the headache and mental

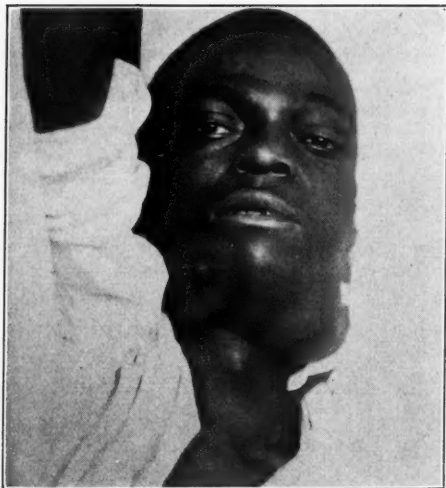


Fig. 1 (Case 5). Showing extensive glandular involvement of both sides of the neck.

symptoms, suggested an intracranial lesion. The condition in the nasopharynx and the small gland in the neck did not seem to indicate a malignant disease of the nasopharynx. This case illustrates the difficulties sometimes encountered in making a diagnosis.

Case 5.—Mr. H. R., aged 24 years, colored, entered Barnes Hospital November 13, 1926, on account of enlargement of the glands on both sides of the neck, generalized pain in the head and difficulty in swallowing. His past history was irrelevant, except that in 1919 he had a Neisserian infection, and in 1920 he had a hard chancre, which was followed by secondary cutaneous lesion. No treatment was taken other than a few pills by mouth.

His illness began two years previously, when he noted a small nodule in the left side of the neck. Soon afterwards a nodule was noted on the right side. These masses rapidly increased in size, particularly during the past year. (Fig. 1.) For five months he complained of severe headache over the right eye, radiating to the side and back of the head. Gradually increasing blindness in the

right eye, accompanied by pain symptoms. For five months he also had increasing difficulty in breathing and swallowing. For two months marked nasal obstruction and pain in the right side of the neck and shoulder had been present. During the two year period of illness there was a weight loss of forty pounds.

Physical examination showed a rather marked recent emaciation. The glands on both sides of the neck in the upper deep cervical regions were markedly enlarged to about 8 cm. in diameter. Eye examination showed an absence of light perception, a slight temporal pallor in the disc and slight impairment of internal rotation in the right eye. The left eye was normal. Partial paralysis of the left seventh nerve. The nasopharynx showed a large, firm, irregular tumor mass almost completely filling the postnasal space. The surface showed superficial ulceration which bled easily. Blood Wassermann negative. X-ray of skull showed nothing definite. Neurologic examination negative. A biopsy of the tumor revealed a highly malignant carcinoma.

Five months later the patient died. Autopsy showed marked infiltration of the base of the skull, paranasal sinuses and metastases to the glands of the neck. The entire base of the skull of the anterior and middle fossæ were involved by the growth. The cella turcica and pituitary body were completely destroyed. It also involved both sides in the region of the greater wing of the sphenoid. The right optic nerve, just anterior to the chiasma, was completely surrounded by the growth, but at no place did it penetrate the meninges. The sphenoids and posterior ethmoids were also involved. The microscopic examination of the tumor showed a carcinoma with large, irregular polyhedral cells with scanty stroma. Many mitotic figures, some areas of hemorrhage and infiltration with lymphocytes and plasma cells. No differentiation.

Comment.—The establishment of the diagnosis in this case was not difficult, because the great enlargement of the glands of the neck and the large primary tumor in the nasopharynx were quite apparent. A remarkable feature of this case was the extensive intracranial invasion of the growth without penetration of the meninges.

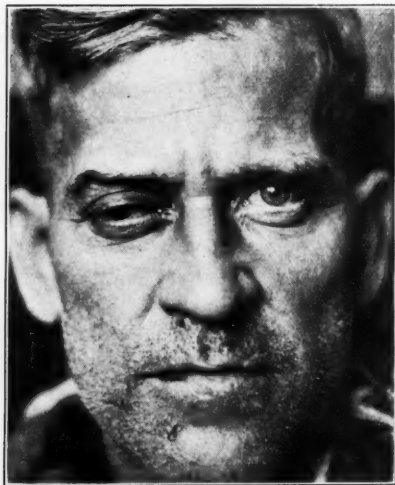


Fig. 2 (Case 6). Complete ophthalmoplegia of right eye with ptosis of upper lid and narrowing of palpebral fissure.

Case 6.—Mr. F. S., age 42 years, entered Barnes Hospital November 2, 1928, complaining of headache, vomiting, loss of vision in the right eye and complete loss of motion of the eye muscles of three months' duration. His symptoms began with headache over the right eye, referred to the side and back of the head. Six weeks before admittance loss of vision was complete in the right eye. His past history indicated that he had had a profuse discharge of pus from the nose six months previously, which he designed as sinus trouble, but this cleared up within a short time. He admitted a Neisserian infection at the age of 17. He also had a chancre and took some antiluetic treatment at that time.

Physical examination showed a complete ophthalmoplegia, slight exophthalmos, complete blindness, sluggish pupillary reaction to light and none to accommodation in the right eye. (Fig. 2.) Right disc appeared slightly pale and margins obliterated. Nutrient vessels faintly seen. Slight edema of both discs. Neurologic examination suggested an early paresis. Spinal fluid showed

14 cells, Wassermann four plus and colloidal gold 23344320000. Blood Wassermann negative, Kahn three plus.

A diagnosis of C. N. S. lues was made, but upon examination of the nasopharynx a suspicious lesion was revealed. The upper posterior part of the septum was absent, and the lower part showed a marked enlargement covered by mucous membrane with enlarged blood vessels. It was bony hard and had the appearance of an osteoma. The vault of the nasopharynx, just posterior to the choanæ on each side, showed small areas of superficially eroded growth with the appearance of granulations. A biopsy taken from the right vault of the nasopharynx showed normal mucous membrane with slight hyperplasia and marked round cell infiltration. About this time the patient developed pain and paresthesia in the second division of the fifth nerve on the right side. Antiluetic treatment was begun and during the subsequent week the eye condition seemed improved. Tissue removed from the nasopharynx again proved no malignancy. Three weeks later two specimens were removed. One section showed areas of fibrosis, necrosis and round cell infiltration with typical tuberculous giant cells. The other section of tissue showed a carcinoma of the transitional cell type described by Quick and Cutler.

Comment.—This case originally presented a very interesting diagnostic problem. The existence of C. N. S. lues immediately suggested the specific cause of the ophthalmoplegia. Even after repeated examination of the nasopharynx, the presence of carcinoma did not seem possible until the patient, while under observation, developed irritation of the second division of the fifth nerve. This very strongly suggested a peripheral lesion. Repeated biopsies were necessary before a diagnosis of carcinoma could be established. Often it is necessary to do this because of the difficulties sometimes encountered in obtaining a section of the growth. The co-existence of lues and tuberculosis is interesting.

Case 7.—Mrs. A. B., age 50 years, entered Barnes Hospital December 8, 1930, on account of enlarged glands in the neck of six years' duration. Small masses on the right side of the neck had been noted for about four years; then they began to grow larger. About a year previous to admission she noted the onset of deafness in the right ear which gradually became more marked.

For nine months she had had headache, earache, pain in the eyes, forehead, right side of the neck and shoulder. During a period of ten weeks she lost forty pounds in weight.

The examination showed a ptosis of the upper lid of the right eye. The right pupil was smaller than the left. The neck showed two hard masses in the upper right deep cervical region, one just behind the angle of the jaw, about 5 cm. in diameter, and another slightly smaller just below it. The examination of the nasopharynx revealed a tumor mass involving the right lateral wall and upper surface of the soft palate. A red area on the palate just above the tonsil appeared to be an extension of the growth. The tumor was firm, irregular and superficially ulcerated.

A biopsy taken from the glands in the right side of the neck showed a squamous type of epithelioma with slight differentiation and many mitotic figures. A biopsy of the tumor in the nasopharynx showed similar characteristics.

Comment.—The unusual feature of this case was the long duration of the glandular involvement. The primary lesion apparently began as a very low grade type of squamous cell epithelioma, which, after a period of about four years, became a very active type of malignancy.

Case 8.—Mr. N. L., aged 17 years, entered Barnes Hospital January 12, 1931, complaining of loss of weight, weakness, epistaxis and vomiting. He had had frequent attacks of epistaxis for twelve years, but for the last year they were much more frequent and severe. He noted tarry stools for one year. Five months before he began to have pain over the right upper jaw and in the teeth, later followed by an aching sensation in the bridge of the nose and eyes. Bleeding from the nose and vomiting of blood became almost a daily occurrence. He then developed pain in the right side of the tongue. He lost twenty-two pounds in weight during a period of six months.

Physical examination revealed marked emaciation and muscular atrophy. A lymph node of rather firm consistency, 3 cm. in diameter, was found below the left angle of the jaw, and a smaller one in the same location on the right. A tentative medical diagnosis of hemophilia and progressive muscular atrophy was made. The neurologic examination was negative except that the pa-



Fig. 3 (Case 8). Lateral view of skull and pharyngeal region showing marked anterior dislocation of air column and large tumor mass projecting from base of skull and posterior wall of nasopharynx.

tient appeared mentally inferior and had a speech defect of the nasal type. Further examination showed a weakness of the internal rectus of the right eye and a ptosis of the right upper lid. The examination of the nasopharynx showed a very shallow cavity, the vault of which was filled with a hard mass, from which bleeding occurred upon the slightest touch. The surface was superficially ulcerated. An X-ray plate of the skull in the lateral position showed a marked anterior dislocation of the air outlining the nasopharynx, suggesting tumefaction at this point with forward dislocation of the pharyngeal cavity. (Fig. 3.)

Under general anesthesia the mass in the nasopharynx was removed by Dr. A. J. Cone, the histologic examination of which showed a very malignant squamous cell type of carcinoma with no tendency to cellular differentiation.

Comment.—The interesting features of this case were the attacks of epistaxis. The nose showed chronic ulcerations and dilated vessels on the septum, which accounted for the nasal bleeding over a period of twelve years. Bleeding from the nasopharynx had occurred apparently for only one year. The X-ray examination of the nasopharynx showed a more extensive growth than was suspected from direct examination. The very free bleeding made it difficult to see the surface of the growth. The involvement of the cranial nerves suggested the existence of a malignant tumor, but the occurrence of carcinoma at the age of seventeen years is certainly unusual. Lymphosarcoma is the type of tumor usually seen in young individuals.

Case 9.—Mrs. S. A., aged 42 years, entered Barnes Hospital on the service of Dr. E. Sachs, January 24, 1931. The onset of her illness began two years before, with intermittent headache over the left temple and orbit which gradually became more frequent and persistent. For a year she noted increasing numbness in the left side of the face and also drooping of the left upper eyelid. In addition there were pain and a sense of fullness in the left ear. Deafness in the left ear was quite marked. For four months she had a slight swelling of the left parotid region. Dimness of vision and diplopia had been present for one year. She lost twenty-five pounds in weight in three months. For six weeks she complained of attacks of palpitation and fluttering of the heart. Dizziness and staggering had been present two months.

Physical examination showed ptosis of the left upper eyelid and paralysis of the third, fourth and sixth nerves. (Fig. 4.) Large venous physiologic cupping in each nerve head. Cutaneous sensation over distribution of entire fifth nerve with corneal anesthesia and loss of taste on the left side. Motor fifth partially paralyzed. Partial paralysis of left seventh nerve. Paralysis of ninth, tenth, eleventh and twelfth nerves on left as shown by absence of taste on posterior part of the tongue, paralysis of



Fig. 4 (Case 9). Complete ophthalmoplegia of left eye. Facial paralysis as shown by drooping of left angle of mouth. Swelling of left cheek from infiltration of tumor.



Fig. 5 (Case 9). Showing paralysis of the left trapezius muscle from involvement of the spinal accessory nerve.

pharyngeal constrictors and soft palate, vocal cord paralysis, weakness of trapezius muscle (Fig. 5) and paralysis of left side of the tongue, adiadokokinesis and suggestion of ataxia in left arm, general hyperactivity of tendon reflexes. Suggestive left Oppenheim.

The examination of the hearing by Dr. C. C. Bunch showed a rather marked decrease almost uniformly for all tones throughout the range, on the left Weber referred to right, and bone conduction decreased on left, suggesting inner ear involvement.

The examination of the nasopharynx digitally by Dr. Sachs revealed a hard mass in the left lateral wall. He asked me to make an inspection, which showed a paralysis of the cranial nerves, as already recorded. Palpation disclosed a very hard mass filling the entire left side of the nasopharynx, extending down to the level of the tonsil. This mass extended laterally into the parotid region and posterior to the angle of the jaw. In the nasopharynx the tumor mass showed a superficial ulceration high up near the vault. The structures of the eustachian tube were invisible.

A biopsy of the tumor taken from the nasopharynx showed a carcinoma which consisted of a mass of epithelial cells with little cytoplasm and large vacuolated nuclei. Moderate amount of anaplasia and mitosis. No keratinization. Lymphocytic infiltration around tumor cells.

Comment.—This patient was referred to Dr. E. Sachs on account of the possibility of an intracranial tumor, but his experience with nasopharyngeal tumors led him to consider this type of lesion in the immediate diagnosis. By palpation with the finger the growth was detected. The extensive cranial nerve involvement as presented in this case is unusual. The extracranial extent of the growth was extremely marked. It had invaded the parotid gland sufficiently to cause swelling of the cheek. It is unusual to find such an extensive primary lesion without marked cervical metastases. The marked reduction in hearing throughout the tone range is interesting. Deafness is generally attributed to involvement of the eustachian tube and is usually of the pure conduction type, showing reduction only for the low tones.

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VII.

THREE UNUSUAL ENDOSCOPIC CASES OCCURRING CONSECUTIVELY.*

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WATERVILLE, MAINE.

These cases might not be classified as unusual in a large bronchoscopic clinic, but to one seeing a relatively smaller number of endoscopic cases what would otherwise be somewhat commonplace may assume the appearance of the unusual. One of them, at least, I do not hesitate to place in this category. All of the three were infants and all of them resulted fatally. That these were seen consecutively, except for some routine endoscopic treatment cases, adds to the uniqueness.

Case 1. Congenital Esophageal Atresia.—This was a baby, six days old, in very poor condition. He had been regurgitating all food taken since birth. X-ray with barium milk showed the esophagus ending in a blind pouch at about the third dorsal vertebra. The stomach and intestines were distended with gas. Esophagoscopy with a 5 mm. tube confirmed the diagnosis. Death occurred the following day from pneumonia.

Postmortem examination disclosed the lungs mottled, with areas of pneumonia throughout. The stomach contained milk curds. The esophagus was dissected out from below upwards and was found to open into the left bronchus just below the bifurcation. The upper segment ended in a blind pouch. The presence of milk curds in the stomach indicated that some of the milk had spilled over into the larynx during attempts at feeding, reaching the stomach by means of the fistula from the left bronchus.

Vogt,¹ in the American Journal of Roentgenology, Nov., 1929, classifies this condition into three types:

1. Complete absence of the esophagus.
2. Those in which both upper and lower segments end in blind pouches.

*Read before Eastern Section of American Laryngological, Rhinological and Otolological Society, New York City, January 9, 1932.



Fig. 1 (Case 1)—X-ray showing upper segment ending in blind pouch, gas bubbles in stomach.

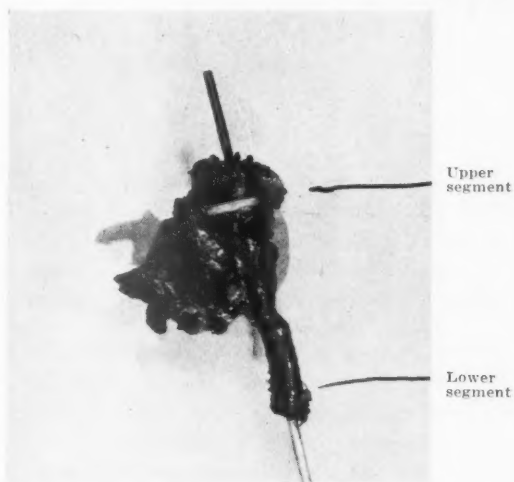


Fig. 2 (Case 1)—Specimen removed postmortem. Looking from behind forward. Upper segment split open. Probe in lower segment passing into trachea and coming out larynx.

3. (a) With fistula between upper segment and trachea.
- (b) With fistula between lower segment and trachea, or bronchus.
- (c) With fistulae between both segments and the air passages.

In types 1, 2 and 3 (a) there will be complete absence of air or gas in the stomach and intestines. On the other hand, in those in which there are fistulae from the lower segment to the air passages gas bubbles will be seen, as in this case. It is perhaps unfortunate that some of the textbooks mention the absence of air in the intestines as one of the diagnostic points of congenital esophageal atresia, ignoring those types with the lower fistulae in which air or gas is present in the stomach.

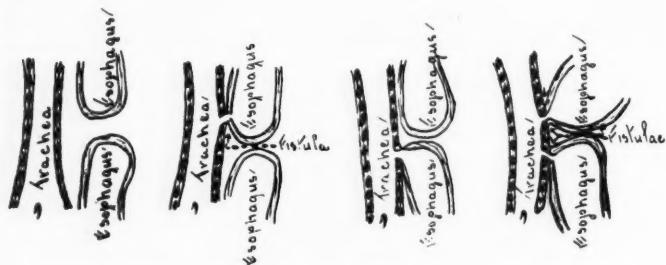


Fig. 3—Diagram taken from Vogt, Amer. Jour. Roent., showing types of atresia.

Case 2. Lye Burns of the Larynx, Trachea, Bronchi and Esophagus.—An eleven months old baby was brought to the hospital with the following history: Seven hours prior to admission the mother was changing its diapers while the baby was on its back on a shelf in the kitchen. A new package of washing powder, just opened, was at hand. Its bright red color attracted the baby, and the first thing the mother noticed the baby was choking and strangling, its mouth frothing, from the powder which it had spilled into it. She attempted to empty the baby's mouth, stating that she pulled out what appeared to be great ropes of rubber-like material. The family physician was called and administered castor oil. Seven hours later the baby was having great difficulty in breathing, so he sent it into the hospital, a

journey of twenty miles. I saw the baby as soon as it arrived. She was breathing with great difficulty, was choked with mucus and there was substernal and intercostal retraction but no cyanosis. The baby's temperature was 103 R., pulse 140, respirations 72. The larynx was immediately examined with a Jackson speculum and large amounts of mucus removed with suction. This was markedly alkaline to litmus. There was marked swelling and edema of the arytenoids and the whole glottis. A small bronchoscope was introduced, and the trachea and bronchi were gently and quickly cleaned with an aspirating cannula. The bronchial mucosa was beefy red and swollen throughout. A low tracheotomy was done while the bronchoscope was still in the trachea.

A pediatrician was called in to direct the medical care of the case. He noted many coarse moist râles throughout the lungs. The baby was given orange juice, which she swallowed without difficulty. The following day the baby was breathing easier, although frequent changing of the tracheotomy tube was necessary. Mucus in copious amounts was frequently aspirated from the trachea and pharynx. While there was more difficulty in swallowing, 15 ounces of fluids were taken this day. Temperature was 105.8. The following day the baby was much worse. It was necessary to change the tube every few minutes. She was not swallowing and fluid given by dropper came out of the tracheotomy tube. Temperature was 108.2. An attempt to pass a feeding tube was unsuccessful.

Direct examination showed the pharyngeal walls, arytenoids, glottis and pyriform sinuses reddened and swollen, with multiple ulcerations throughout. An esophagoscope was passed to the stomach. Ulcerations were noted throughout the esophagus and on the rugæ of the stomach. A small feeding tube was introduced and the esophagoscope withdrawn. The baby expired shortly after this, all attempts at resuscitation failing. While no post-mortem was allowed, both the esophagus and air passages were carefully examined endoscopically after death. Ulcerations were found throughout the trachea, bronchi and esophagus. No tracheo-esophageal fistula could be found.

This was a real tragedy and brings vividly to mind the oft repeated pleas of Jackson² and others for the need of legislation

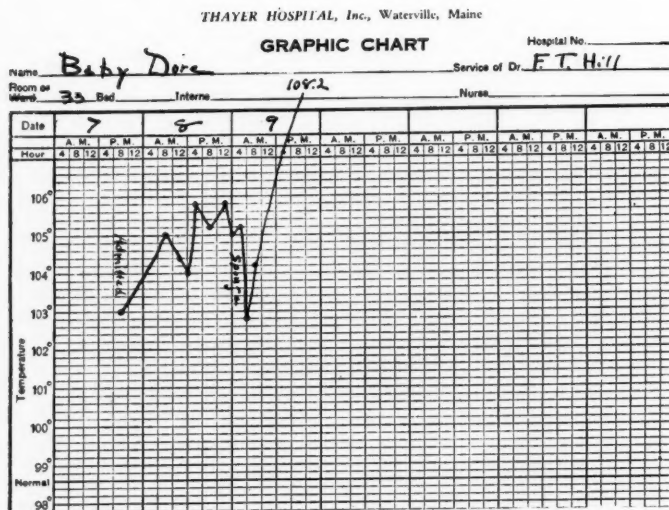


Fig. 4 (Case 2)—Temperature chart.

stipulating that such commercial preparations shall be labeled so as to denote their dangerous character if aspirated or swallowed. This popular powder carried on its package the words "Does not injure skin or fabrics" and that it contained "no lye, salsoda, soap, grit, ammonia, oxalic acid, caustic or animal fat." A sample of the powder was analyzed in the department of chemistry, Colby College. Quoting from the report we find that, "The aqueous solution is basic to litmus, a suspension in absolute alcohol is basic to phenolphthalein, indicating the presence of free NaOH. Hydrolysis accounts for the basic nature in aqueous solution." Analysis showed the composition to be—

- Na_2CO_3 (sodium carbonate)
- $\text{Na}_2\text{B}_4\text{O}_7 \cdot 10\text{H}_2\text{O}$ (borax)
- $\text{NaBO}_3 \cdot 4\text{H}_2\text{O}$ (sodium perborate)
- $\text{Na}_3\text{PO}_4 \cdot 12\text{H}_2\text{O}$ (sodium phosphate)

As the sodium carbonate was not present in large quantities it was hard to say whether it had been introduced intentionally or was present as an impurity.

In a communication accompanying the report of analysis, Dr. George F. Parmenter, professor of chemistry at Colby, said: "You will note that sodium perborate is one of the compounds found. Whenever this substance comes in contact with water it is hydrolyzed and it acts not only as a strong oxidizing agent but the solution is strongly alkaline. The sodium phosphate also forms on hydrolysis an alkaline solution, as do the other two compounds, borax and sodium carbonate. A mixture of these compounds produces a very active washing powder which should be used only where an active detergent is required."

It would seem that the label on the package was deliberately misleading, as free NaOH was formed in large quantities by hydrolysis as soon as the powder went into aqueous solution. Since the hydroxide is highly dissociated there is produced a solution which is very caustic. This was obviously far too dangerous a preparation to be left around where children might get hold of it. At least a warning of its possibilities, if taken internally, should have been included on the label, as well as a suggested antidote. Were this the case the mother might have been able to have rendered more efficient first aid, and the family physician might have given more effective treatment than castor oil. Of course, the delay of seven hours before seeking more efficient aid was unfortunate. The widespread involvement of the mucosa throughout the air and food passages, noted at that time, was significant of the destructive character of the chemical. Possibly the insertion of a feeding tube when first seen might have been beneficial, but the baby seemed to be swallowing very well after the passage had been aspirated. She was in no condition to stand gastrostomy.

Case 3. Rupture of the Trachea at Delivery.—To the best of my knowledge, this case is unusual. A male baby, weighing 11 pounds, was born after a very difficult labor of 20 hours. The mother was a para 111, 32 years of age and weighing 201 pounds. Her next youngest child was ten years old. Her obstetrical history was reported as negative. She was delivered at home by a competent obstetrician of experience, who had served as resident in one of our largest metropolitan maternity hospitals. He had the assistance of a competent nurse. According to the obstetri-

cian's records, the mother's measurements were essentially normal—spines 25 cm., crests 31 cm., external conjugate 23 cm.; although the outlet measurements were possibly small, being $9\frac{1}{2}$ for the transverse diameter and $12\frac{1}{2}$ for the anteroposterior. The internal conjugate was not obtained, as the head was low when seen. The baby was in a R. O. P. position. The head came down and rotated slowly, being delivered without instruments. Ether anesthesia was given. There was great difficulty with the aftercoming shoulders, which were extremely broad. These were held up by the bony pelvis and the doctor was obliged to use a great deal of force to deliver them.

After delivery the baby showed a great deal of difficulty in breathing. Usual measures to relieve this were without appreciable effect. Apparently there was no expansion of the left lung. Believing that he had a case of asphyxia of the newborn, the doctor called me in consultation about two hours later. When I saw the baby he was cyanotic and the breathing was hardly perceptible. The neck was greatly swollen and somewhat crepitant, but at that time this feature did not present the significance it should have had to me. The larynx was examined with the speculum and appeared normal. A Tucker 3 mm. (newborn) bronchoscope was introduced, and on examining the trachea a horizontal tear was found on the anterior wall at about the level of the third ring. This was more easily seen as the doctor, who was using artificial respiration, compressed the thorax. The right main bronchus seemed clear, but blood and mucus were found and aspirated from the left bronchus with a small cannula. As the heartbeat had now ceased, nothing further was attempted. In the forcible extraction of the shoulders, occasioned by their disproportion to the maternal pelvis, the trachea had been torn and ruptured with resulting emphysema of the tissues of the neck.

It was unfortunate that this had not been a hospital case, as possibly some operative procedure, such as either a pubiotomy or fracturing of the baby's clavicle might have allowed the delivery of the shoulders without this trauma. Had the outcome been the same, there would have been a much better likelihood of obtaining a postmortem examination. For some reason or other, it is more difficult, usually, to get this permission in the home. I

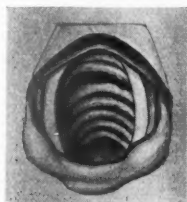


Fig. 5 (Case 3)—Drawing showing tear in anterior wall of trachea.

assume that death was caused by asphyxia. Certainly its appearance suggested this diagnosis. Of course, efforts at artificial respiration increased the emphysema and thus defeated its purpose. I have considered the possible effect of tracheotomy had the condition been recognized earlier and have wondered if, after all, this would have been worth while. At any rate, this might be an argument for hospitalization of all obstetrical cases so that any such emergency might be handled more intelligently and efficiently.

CONCLUSIONS.

Three infant endoscopic cases are presented. The first merely suggests the need of some slight revision in textbooks regarding the diagnosis of congenital esophageal atresia. The second emphasizes the need of protection of the public by proper labeling of "washing powders," warning of their dangerous character if aspirated, and of further education of the family physicians that they may better realize the possibilities of such accidents. The third suggests the advisability of hospitalization of obstetrical cases, that any such emergency can be more efficiently managed, and brings up the question of tracheotomy in such a case as above reported.

PROFESSIONAL BUILDING.

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VIII.

BLOOD COUNT, METABOLIC RATE AND BLOOD PRESSURES IN NASAL SINUSITIS.*

EUGENE R. LEWIS, M. D.,

LOS ANGELES.

The observations herewith presented are based on a review of 1500 consecutive cases of sinus troubles, in whom studies have been made of basal metabolism, blood counts, body weight, blood and pulse pressures at clinics of the Los Angeles General and White Memorial hospitals and in private practice. These patients had been treated previously, surgically and nonsurgically. In approximately 10 per cent of the cases there was coexistent eye trouble, such as conjunctivitis, keratitis, anterior and posterior uveitis and hypertension. Ocular tensions taken in many cases revealed unsuspected tension abnormalities, but statistical analysis of abnormal ocular tensions cannot be made because of incompleteness of many records. Information previously charted on the records of these cases included notes describing nose and throat and radiograph findings, operative and nonoperative treatments, with occasional urinalysis, blood count and coagulation tests. My observations were undertaken in order to look into the nature of causes of local pathology and the significance of relations between systemic and local conditions found in these cases. For a number of years I have been increasingly convinced that there are definite meanings, resolvable into clinical values, to be deciphered from frequently recurring, consistent relations between laboratory findings, local findings and general clinical findings in sinus disease. Insofar as these findings tend to group themselves into recognizable pattern, hitherto largely meaningless jumbles of clinical and laboratory data assume commensurately more coherence and intelligibility. This pattern reflects the general nature of biochemical and biophysical changes in living matter observed as clinical phenomena. Whatever steps can be taken to reduce

*Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, January 29, 1932.

physiology, pathology, chemistry and physics to common terms tend to make them of greater value and use in solving medical problems.

In reviewing these cases the following items stand out with special prominence:

1. Mendelian transmission of dominant characteristics.
2. Instability of fluid distributions in body tissues.
3. Habitually low fluid intake.
4. Low protein and fat intake.
5. Relatively high color index.
6. Abnormal incidence of band forms, large mononuclears, basophiles and eosinophiles and relatively low lymphocytosis.
7. Labilities of cardiovascular pressures.
8. Low basal metabolic rate.
9. Abnormal texture and color of mucosa, regardless of infectious activities.

1. Family history shows father-to-daughter and mother-to-son transmission of tendencies toward the clinical troubles under discussion. Often the data seem to show the reverse of this heredity, only to lead to the discovery of hitherto unrevealed dominant strain in the heterologous parental line. Only careful checking and rechecking of data will establish these facts.

Among significant items identifying such heredities are hay fever, asthma, allergies, "catarrhal" troubles, spasmophilia of all kinds, recurrent pyelocystitis, epilepsy, pruritic skin troubles, "dyspepsia," chronic colitis, hives, eczematous skin troubles, stone and tartar formations, hyper-ceruminosis, unusual weight, arthritis, tendencies to synovial or bursal effusions, essential circulatory hypertension, ocular hypertension, vernal catarrh or seasonal conjunctivitis.

Among significant items of previous personal history, colic, skin troubles, spasms in infancy; susceptibilities to colds and rashes, croup, pyelitis, hives, growing pains, chilblains and food incompatibilities in childhood; migraine, epilepsy, rheumatism, so-called "neuralgias," appendicitis, gastric ulcer, spastic colon and colitis, hay fever, asthma, mucoid or purulent catarrh, tracheitis, bronchitis, eczema, pruritis, leukorrhea, urticaria, angio-neurotic edema, cystitis and arthritis.

Important items of present history include onset details, progress and recurrences, seasonal and geographic details; individual food and drink habits; disturbances of digestion and bowel action; weight variations; abnormalities of sleep and thirst; itchings of all kinds (scalp, ear canals, conjunctivæ, palate, skin), leukorrhea, early morning to midday onset of headaches and spells of sneezing, nasal obstruction and discharges from nose or throat; incidence of tonsillar, otitic or laryngeal complications.

In my opinion, an accurately completed history is at least equal in value to the ordinary examination of the upper respiratory tract including radiographs.

Inheritance of the tendency to troubles in this category is definite; it is suggestive of abnormal cellular catalysis and appears as a dominant characteristic. It is frequently possible to supplement items elicited in the history by physical findings in other individuals of the family. The discovery of evidence in a son of such troubles known to exist in the father (or in mother and daughter) has frequently cast doubt upon the soundness of this conception of heredities. These apparent inconsistencies, however, have frequently led to the revelation of unsuspected dominant strain in the other parent.

2. Unusual fluid movements in the tissues may be revealed as:
(a) Facile changes in weight, often amounting to 2 per cent or more of total body weight in 24 hours or less. On several occasions it has happened to be possible to detect sudden variations of four or five pounds within 24 hours (one patient was under her physician's observation while she lost seven pounds in two hours.)

(b) Sudden change in bulk of body areas, puffiness of loose areolar tissue especially noticeable in the eyelids (also found in the extremities after unusual walking, standing or pressure); blister formation caused by minor degrees of heat or friction; sudden blocking of one naris changing—in a minute or less—to complete unblocking and coincidental blocking of the opposite naris.*

*This blocking is easily differentiated from turgescence of erectile tissue in that it occurs in areas where erectile tissue does not exist. Further differentiations may be made by inducing turgescence by local mechanical irritation of turbinates and comparing the findings with those seen in blocking.

(c) Discharge of watery fluids, as vomitus or diarrhea unassociated with gastro-intestinal disturbances, as sialorrhea, lachrimation or rhinorrhea without concomitant evidences of irritation in salivary, lacrimal or nasal areas; from a half to two teaspoonfuls of watery fluid may gush from one nostril (sometimes termed "nasal hydrorrhea," or "discharge of cerebrospinal fluid from the nose"). This is very commonly observed after swimming.

(d) Abnormal fluid condition of mucous membrane: when desiccated it is red, thin, parched and friable; when saturated it is pallid, pebbled, milky, with or without edematous polyps. Sudden discharges into the nose—watery, mucoid or mucopurulent—may occur upon parched mucosa as well as upon hydropic mucosa; at other times either type of mucosa may show little or no secretion upon its surface.

3. Abnormally little thirst is experienced by a large number of subjects; this is more apparent in adults than in children, but is characteristic of the condition, regardless of age. "Secondary thirst," resulting from mouth breathing, is often found. This is easily differentiated from bodily (or tissue) thirst.

4. Almost without exception these subjects eat relatively little protein and fats, and careful questioning often reveals such dietary habits extending to early years of childhood. In adults abstention from proteins may be increased by erroneous impressions—that they are not good for "full-blooded" people or are bad for "high blood pressure"—that fats cause obesity, liver troubles and intestinal indigestion; that milk, cream and butter are "mucus-forming" foods.

5. The color index is by no means always abnormally high in these cases. Nevertheless, in reviewing blood findings attention is attracted to the frequency with which relatively high color indices appear; and clinical improvement with increased red cell count is frequently associated with noticeable lowering of high color index.

6. Leukocytosis seems to have shown no consistent level in these conditions (except that it rises during acute infectious phases), nor does the polymorphonuclear percentage; band forms in 2 to 5 per cent, lymphocytes between 10 and 15 per cent below

average, large mononuclears, basophiles and eosinophiles commensurately above the average, are almost universally found. These differences, in general, characterize the hemogram.

7. Cardiovascular pressures are outstandingly abnormal with respect to systolic level, pulse pressure and stability. High systolic level is the rule, regardless of age; it is not uncommon to find systolic pressures approximating 160 between the ages of 12 to 16 years, commensurately higher with increasing years, up to 200—250 mm. Hg. In the young a more changeable systolic level is found; pulse pressures on initial examinations are typically high regardless of age. In general, cases show alterations of both systolic level and pulse pressure; and quiescent stages show high systolic levels and pulse pressures regardless of age.

8. Basal metabolic rate was almost universally low, without definite relation between body weight, blood pressure and metabolic rate; high systolic level is not significant of high metabolic rate, and is frequently found in association with low metabolic rate. Body weight is more frequently above the average than below it; but low metabolic rate is found in association with facile weight changes, both up and down.

9. The mucosa is often hydropic—soggy, pebbled and light colored. This is true particularly of the mucosa on the lateral nasal wall, although it is occasionally found in the septal mucosa also. At times, however, it may be red—even parched and friable—in certain areas or throughout the nose.

The etiology of these conditions is primarily systemic; local effects are manifested in nasal areas as sinus trouble, in vaginal areas as leukorrheas, in the large bowel as colitis, in bladder-ureteral areas as pyelitis, in skin areas as pruritis or eczema, intracranially as chorea, migraine or epilepsy. Allergy, in the sense of "specific foreign protein sensitivity," may be demonstrable at times in such cases, but cannot be regarded as the ultimate cause. Blood pressures and tissue tensions, metabolic rates and hemocytologic findings reveal important underlying constitutional conditions; in addition to local examinations, collateral evidence concerning these factors is indispensable to full knowledge of the condition and progress of an individual case. As body chem-

istry changes, osmotic pressures and tissue reactions change; nutritional and fluid metabolism and hormone balance reflect these changes; local symptoms and lesions constitute far less than the entire clinical picture. These cases often go on for months or years with no evidence of infectiousness, even when pathogenic bacteria are present. These tissues may show bacterial infection at times. The extent and intensity of infectious processes depend upon the extent to which chemical and other conditions of tissues undergoing bacterial permeation prove favorable to their culture and dissemination. Bacterial factors in general must be regarded as secondary; primary etiologic factors concern body chemistry. The nature and causes of altered body chemistry are various; catalysts are assuming aspects of increasing importance as factors in etiology; nutrition (in its broadest sense) has proven in my experience to be a very important and tangible factor. By definitely controlled variations of the entire intake of the body it has been possible to precipitate attacks of sinus troubles and other allied conditions; by reversing such control, to cause such attacks to subside.

The routine use of serial observations of tissue tensions, body weight, basal metabolic rate and hemograms in conjunction with the usual methods of examination and the routine study of tissue reactions to chemical alterative measures are of prime importance in diagnosis and treatment of nasal sinus troubles.

1154 ROOSEVELT BUILDING.

IX.

REACTIONS TO INFECTION.*

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Formerly it was upon clinical observations and the deductions made therefrom that progress leaned most heavily for its support. Today the research laboratory, first with bacteriology and now with biochemistry, has deservedly established its position. The literature from this source has developed a nomenclature that often leaves a mere clinician bewildered. The desire to clarify in my own mind some of the modern conceptions as to human reactions to infections led me to undertake this review.

Clinical experience has demonstrated the changes that take place in the formed elements of the blood during the course of and recovery from an infection. Metchnikoff was without any knowledge of the humoral aspects of immunity, yet he recognized that recovery from streptococcal infections seemed to depend upon the presence of mononuclear cells and he termed them macrophages. The red corpuscles, the blood platelets and the leucocytes constitute the formed elements of the blood. Blood serum with its cellular elements, is not a special form of connective tissue, but rather the product of activities on the part of body tissues. Since the cellular elements are not formed in the blood stream, diseases of the blood *per se* do not exist, but diseases of the blood forming organs do occur. Thus it becomes of prime importance to know the source of the cellular elements of the blood. To the blood forming tissues has been given the name, the hematopoietic system. Since the leucocytic changes predominate in infections, the red corpuscles and blood platelets will only be incidentally considered.

The embryologic history of the blood immediately brings one to the controversy regarding the postnatal origin of blood cells.

*Presented before the Mid-Western Section of the American Laryngological, Rhinological and Otological Society, Kansas City, January 21, 1932.

One school, the polyphyletists, opposed to the monophyletists, claim that before birth the common ancestral type for all cells disappears and that only specific stem cells remain for each type of cell.

The terminal syllable blast is applied to the earliest form of the cell in its developmental history. The embryologic type of red cells, entodermal in origin, first seen with a nucleus, is known as a megaloblast and has a giant non-nucleated offspring known as the megalocyte which is not represented in postnatal life. Their presence occasionally after birth has, to some, indicated a pernicious anemia, but a more commonly accepted theory is that it is because of a retention of prenatal tissues. Postnatally, in the order of development, there is recognized the macronormoblast, nucleated and without hemoglobin, then the normoblast, nucleated and containing hemoglobin, which precedes the non-nucleated blood cell.

The leucocytic group is divided into two general groups, granular and nongranular, according to the appearance of the cell cytoplasm. The granular group includes the polymorphonuclears and the monocytes; the nongranular group, the lymphocytes. The polymorphonuclears are subdivided, according to the staining propensities of the cytoplasm, into neutrophiles, eosinophiles and basophiles. Ancestral types of the three classes of polymorphonuclears follow a similar developmental history and are known in order of ancestral age as the myeloblast (nongranular), premyelocyte and myelocyte (granular, with round nuclei and present only in pathologic conditions); and the metamyelocyte (with a bent or indented nucleus) and occasionally found in normal blood. The monocyte, granular, larger than the polys, differentiated by certain staining qualities, preceded ancestrally by the promonocyte, has an origin which is not entirely clear. Lymphocytes are nongranular, the majority being small and distinguished by a protoplasmic rim. They develop in the follicles which are formed in connection with the lymphatic channels. In the embryo, besides the purely hemoglobinous cells from the entoderm, there are collections of mesenchyme from which red and white cells develop. From this mesenchymal mass develops the connective tissue framework, the blood and lymphatic vessels. Most of the

fixed cells become fibroblasts—that is, collagen producing cells, but with them there remains a variable amount of wandering cells of a monocytic type. These are wandering cells among the fixed cells. They are undifferentiated but distinguishable by vital stains and are reservoirs with fibroblastic or blood cell potentialities. They are known as macrophages, rest wandering cells, clasmotocytes and histiocytes. In the spleen and marrow, of which we think as the source of our blood cells, they form continuous masses closely connected with the reticular framework. These cells have been shown to have markedly phagocytic qualities, but this, of course, is only a property which all cells have to a greater or lesser degree. These cells, which are to be distinguished, first, from connective tissue cells and then from myeloid and lymphatic cells, include many cells of the splenic pulp, of medullary follicles of the lymphatic glands, of other lymphatic structures, of the splenic sinuses, of the liver capillaries (stellate cells of Kupffer), of the capillaries of formative bone and of the adrenal and pituitary. They retain a primary avidity for phagocytosis. In addition, they have a selective storage capacity and extraordinary regenerative powers after injury.

As to their selective capacity, injections have been made with two different dyes (Schuttenhelm), and some cells will contain one dye and some the other, with very little mixing of the two. Remnants of what are considered partially digested red cells have been noted in these cells in the splenic pulp. Around extravasations of blood they have been found to contain ferruginous pigment, evidence of the power to take up diffused hemoglobin. Their importance has been further emphasized experimentally to substantiate the claim that particularly do these cells, so intimately associated with the lining of the vascular channels of marrow, take the place of the spleen after its removal in the process of blood destruction or erythrophagocytosis. The liver cells have shown an inability to, by themselves, extract liberated hemoglobin from the plasma following a hemolytic poison, when the reticulo-endothelial cells have had their powers impaired by a sufficient amount of dyes. If, as Rich has claimed, altered hemoglobin is the only source of bile pigment, then these cells seem to play an important part in the metabolism of hemoglobin. Endocrine

stimulation affects the storage capacity of this system of cells; that for trypan blue is increased by insulin and decreased by thyroid extract.

While normally blood cells develop mainly by a cell division, there must be source for new cells. It has been shown that under stress of a sufficiently long continued stimulation of the reticulo-endothelial cells there takes place a myeloid metaplasia—that is, the formation of red cells and leucocytes in places where they are normally absent. It is acknowledged that myeloid and lymphatic cells may arise from the reticulo-endothelial cells, and Piney claims this mesenchymal structure, or rather cell, is the only polyvalent blood producing cell—that is, a hematopoietic cell.

It is claimed by some that the monocyte is a direct derivative of the reticulo-endothelial system, and perhaps a clinical verification is seen in rare conditions of systematic overgrowth of the reticulo-endothelial system, the leukemic blood picture showing the monocyte as the predominating cell type. Long has shown the development of granules in some and hemoglobin in other reticulo-endothelial cells, and experiments tend to show that from the identically same type of parent cell occurs a process of erythropoiesis, red cell formation takes place intravascularly and leucocytic formation takes place extravascularly.

Infections ordinarily are accompanied by a leucocytosis, the type depending on the addition of neutrophilic granular cells, reduction of eosinophilic cells and possibly the monocytes. Arneth first called attention to the greater importance of the qualitative rather than the quantitative change in the type of circulating blood cell. Cook and Pander simplified his complex classification by differentiating those parts of the nucleus that are connected only by a chromatin filament. Then Schilling gave his so-called band cell differentiation with the shift to the left or right, which has been accepted in clinical work more than any other.

It would seem that the parent tissue, the reticulo-endothelium, becomes actively engaged at one time in the production of one type of cell at the expense of the others. Environmental conditions produce different types of cells and for long periods of

time it does not seem possible to continue a normal number of red cells with an excess of leucocytes, which explains the anemia accompanying a myelosis.

Clinically we commonly measure the degree of infection present by the blood picture—that is, by the degree of hemoblastosis, a term including all forms of hematopoietic excess. Since the pathology is primarily to be applied to the blood forming organs and since we have atypical reactions, impossible of classification, it is to be remembered that an increase in the number of immature cells is not simply because of an excessive destruction of cells with an inability of the cellular forming tissues to supply adult cells in proportion to the demands of the body. Infections are generally accompanied by a leucocytosis and, in the progressive stage, characterized by a neutrophilia (increase of the neutrophilic granular cells) and a reduction of the eosinophilic with, at times, also the monocytic cells. During the postinfective stage one interprets the condition according to the neutrophilic decline, increase of lymphocytes and also of the eosinophiles and monocytes. While a lymphocytosis, in the postinfective stage, is a sign that the infection has been overcome or at least has been held in check, it must be remembered that it usually accompanies but is not strictly an indication of chronicity. A lymphocytopenia during the course of a persistent infection is a bad sign. A sudden fall in the number of lymphocytes together with a progressive neutrophilia is an indication of an extension of the inflammatory process. A lymphocytopenia with a progressive decrease of total leucocytes and a shift to the left is a very serious sign. With an additional absence of eosinophiles one has the typical picture seen in moribund patients.

The function of the eosinophilic cell is not understood, yet its presence is not without significance. Eosinophilia of mild degree is usual in disorders of the internal secretion functions. Such disorders may also cause a monocytic increase even up to 15 per cent. An increasing eosinopenia with progressive leucocytosis indicates a progressive infection and, if present with a falling leucocytosis, may mean that the infection has overpowered the defense mechanisms of the body. The reappearance of eosinophilic cells is always a favorable sign, and with a lymphocytosis follow-

ing a neutrophilia and a shift to the right, one has clinical indications of recovery. The reappearance of eosinophiles with a falling neutrophilia is an indication toward recovery.

It is the correlation of these clinical signs, a few of which have just been given, to the hematopoietic system that offers the greatest opportunities for advancement in combating infection and acquiring and maintaining a state of immunity.

The development of the human organism is from a single cell. Differentiation follows in lesser or greater degree as we advance in the scale of life. The fact that certain cells in the higher forms of life show special ingestive or phagocytic ability is only evidence of the retention of a cellular function necessary to life in the lower forms. Here digestion is primarily intracellular, foreign substances being ingested, broken down and utilized as food. As we ascend in the scale digestion becomes largely extracellular. Enzymes appear that render food materials diffusible and chemically adjusted to cellular needs. The fats become fatty acids and glycerin, the proteins the amino acids and the carbohydrates the sugars.

When Metchnikoff, in 1882, noted the phagocytic action of certain cells, the science of immunology was born. Then Von Behring and Kidasato, in 1890, and Buchner, in 1891, called attention to the humoral or fluid aspects of the circulating fluids of the body.

Disease from extraneous sources recognizes two fundamental variables, the virulence of the invader and the resistance of the host, and its presence is manifest by the reaction between invader and host. This interaction on the part of the host takes place through the blood plasma, certain nucleated cells and certain fixed cells.

Unaltered fats and carbohydrates in the circulation do not seem to have any great influence on immunologic problems. Against the proteins the body marshals its defense forces by neutralizing poisons or actually destroying the invader. Bacterial cells as complete proteins are not essentially toxic to body tissues.

Bacteria differ from other foreign proteins in that they are living organisms. The majority are economically independent,

being able to live on dead organic matter. Fortunately for us, only a small number, relatively speaking, have developed the power to live in and upon the animal body. From being pure saprophytes they may adapt themselves to the exterior body surface, living on excretions and waste products. Again, they may exist on the exterior, yet become pathogenic by elaborating an absorbable poison, as in diphtheria and tetanus. Further, they may actually invade the tissues, to which class belong most of our pathogenic bacteria. Finally they may be strictly pathogenic to certain species or under certain conditions prevailing in the body. The variations in adaptability of bacteria have been amply demonstrated in the laboratory as to virulency. What will come from the studies in bacterial mutation one cannot state except that they promise to establish facts that will be of the utmost importance.

The reaction of the host to invading micro-organisms varies from an absence of proteolytic substances, that permits a generalized infection before a reaction is induced, as with the *spirocheta pallida*, or an actual destruction of the organisms to which the body has been constantly exposed, without any general morphologic reaction, as with the *staphylococcus*. Again, the entrance of the organisms may be in such numbers or of such virulency as to overwhelm the host before the defense forces can be mobilized.

It is the correlation between the cellular and the serologic elements that has disturbed the science of immunology. The prominence given the humoral elements following the announcement of Ehrlich's side chain theory, which has always remained a theory and yet not to date entirely disproven, has to answer many discrepancies in order to justify itself.

First consider the term antigen, which is purely functional, is not confined to bacteria but to any substance which can arouse tissue reaction in a specific way. Reacting to the presence of an antigen there may appear in the humoral elements of the tissues a chemical rearrangement, without structural identification, yet functionally a definite substance which reacts specifically to a given antigen. That it has substance has been proven in the laboratory by its absorption from an immune serum by bacteria and

the further recovery of this specific reacting substance from the precipitated bacteria. To this substance is given the name antibody.

The various antibodies are named according to the resulting antigen effect, as antitoxins, lysins, tropins, opsonins, agglutinins, precipitins and anaphylactic antibodies. Sera or plasma from normal animals has antagonistic properties to bacteria and so, as far as known, antibodies represent an increased physiologic function. The absence or presence of antibody-antigen reaction in the test tube is no measure of the efficacy of serums therapeutically or prophylactically. It is an established fact that the antibody reactions of most limited value therapeutically are of the greatest value diagnostically. This is demonstrated in the following reactions: the agglutinins in typhoid fever, the complement fixation in syphilis, the precipitins in anaphylaxis and possibly certain allergic reactions.

Demonstrable antibodies in the test tube may have no relation to the presence or absence of the immunity in the individual. Antibodies may be entirely absent in the serum of a patient who has recovered from and is most highly protected against a disease, as after recovery from typhoid fever. Antibodies are present in smaller amounts in the actively immune individual than in one who has been immunized artificially by serums. Antiserums directed against bacteria themselves have been failures except for that for the meningococcus, possibly the bacillus tetanus and, to a limited extent, the pneumococcus.

Laboratory experiments have increased the effectiveness of antibacterial serums by cell stimulation, by the addition of cells to the serum and by the addition of cellular exudates from an immune animal, whereas the addition of exudates from normal cells was without effect. The susceptibility of bacterial antigens to the humoral elements of the host is increased by absorption by the antigen of the antibody so that it becomes vulnerable to another seral constituent known as the complement of alexin. This constituent is markedly thermolabile and deteriorates on standing and possesses enzyme-like properties—that is, solvent as well as destructive powers.

Since the absence of antibodies in the presence of recovery and the failure of antibacterial sera are known facts, one must turn to some other factor as the predominating influence in the host's defense mechanism against infection. Clarke and Gay have suggested that the ineffectiveness of the antibodies is due to lack of tissue response—that is, a failure on the part of the cellular elements to complete the tropinizing (phagocytic) effect.

Cellular activities are of prime importance, forming the constant factor necessary in overcoming infection. Antibody and antitoxin formation can only be explained on the basis of cellular activity. Chemical transformation, except with antitoxin, does not explain the usual method of recovery, first because the production of antibodies is out of all proportion to the antigen, and secondly, because protection is afforded without the presence of antibodies. Antibodies are produced not to directly overcome infection or to maintain protection, but rather to facilitate the destruction of the antigen either by a normal constituent of the humoral element of the blood, the alexin, or by the cellular elements of the blood and by certain fixed tissue cells. Thus by elimination, cellular activities, first by the production of definite chemical substances, prepare antigens for their destruction by the major cellular function, that of phagocytosis. This is the physiologic cellular function retained to some extent by all cells as an ancestral inheritance, but with avidity by the myeloid circulating elements of the blood and certain endothelial and so-called reticular cells. Since it has definitely been proven that the cells of the reticulo-endothelial system not only retain a primary avidity for phagocytosis, but also are able, according to environmental conditions, to produce the various cellular elements of the blood, their importance in immunology and overcoming infection becomes of primary interest. The specificity of cellular antigen reaction remains one of the mysteries. The production of neutrophilic elements in combating infection may be likened to a first line of defense. They are often inadequate. In the case of streptococcal infections, lack of antibody response has been given as the cause—that is, a failure on the part of certain cells to produce a definite chemical substance. With recovery from streptococcal infections there is usually an increase of monocytic

cells. The appearance of eosinophilic elements is an indication of a change toward recovery. It is as if the barrage laid down by the neutrophilic artillery requires a mop-up squad to accomplish recovery. What are the environmental conditions that vary the proportion of untrained combatants in the first line of defense (immature cells), and why are these immature combatants less capable than more mature members?

Why should not the immature elements have a greater phagocytic power, since they are nearer their ancestral type which has as a primary function phagocytic ingestion and digestion of heterologous substances? As the scale of life advances, physiologic cell specialization occurs. Experimentally cell selectivity has been demonstrated among the cells of the reticulo-endothelial system. How far does this go and what causes this system of cellular structures to produce certain cells in excess at one time and to change to another type during the defense offered to an extraneous enemy? Will the secret of cell specificity to antigen reaction, or will cell specificity as to phagocytosis give the information by which infections will be universally rendered impotent? These are some of the questions that confront the problem at the present time.

The invasion of the tissues by bacteria differs from other proteins because they are themselves living organisms. They constitute a living antigen that can multiply at the expense of the host with possibly specific toxic possibilities and specific localizing powers in the host's tissues. Inflammatory responses and the resulting phagocytosis due to cellular activity in the naturally immune may go on independent of antibody formation.

The protection afforded the individual by the formation of granulation tissue is brought about in an entirely different way from an antigen-antibody reaction. It is here that there is developed to an excess amount the so-called histiocyte or clasmotocyte products of the reticulo-endothelial system, which by their primary phagocytic powers destroy the invader. Wilensky has shown that the phagocytic powers of the cells of the reticulo-endothelial system are markedly reduced for the dyes during an infection. Urbach and Schnizler have shown that the injection of tumor cell

emulsion was ineffective until a preceding injection of India ink had been given. Such experiments give a prominence to the part that the reticulo-endothelial cells play in immunity, but to completely explain their function is difficult because of the inability to experimentally completely abate their functional activities.

Infection, as represented by the entrance into the body of heterologous protein material, meets with resistance by other factors than those under discussion. The skin, acidity of the gastric juice, differences in species as well as racial and individual, the body enzymes, the liver and the endocrine system, are some of the factors that play their parts. However, the limits of time prevent their discussion here.

When infection occurs, the accumulation of cellular elements at the site of the infection is evidence of some governing force. The laws of chemotaxis which govern that inherent but mysterious force between substances, to either positively attract or negatively repel one another, apply to infective agents. Copper, mercury, living bacteria, dead bacteria, bacterial proteins and the products of tissue destruction act positively to leucocytes. Gold and iron act negatively to the white cells.

An antigen is a nondiffusible protein material which has the power of exciting a specific response. Bacterial antigens are but one of the group but a very important one. Here we have a complex molecule but with a fractional part. This fraction has been demonstrated to be a carbohydrate radical attached to the protein molecule and spoken of by Ehrlich as the haptophore group, by Landsteiner as the haptene fraction and by Zinsser as the residue fraction. This specific fraction will react with the antibody but cannot induce antibody formation.

Antigen possesses a mysterious power to elicit by contact a specific change in cellular reaction capacity. Bacteria do not differ from other proteins in this regard. This changed cellular reaction capacity may lead to the formation and release from the cell of free antibodies. Circulating or free antibodies are important but constitute an incidental rather than a fundamental difference from the antibodies that exist in an intracellular content.

The tissue cell is the functional unit that serves as a source of the various protective constituents of normal and immune sera.

The exact location of these antibody forming cells and tissues is not definitely proven. It seems self-evident that it must take place throughout the body, and this has suggested the likelihood of local immunization as advocated by Bezredka. In the last few years the reticulo-endothelial system, particularly the endothelium of the capillaries, has been considered as the most probable source. Aschoff emphasized that it fulfilled the necessary wide distribution, and Gay pointed out that it includes the most markedly phagocytic cells of the body.

With an infinite number of antigens it seems incomprehensible that the cell could contain enough specific receptors. Manwaring first questioned this phase of Ehrlich's theory. There is, of course, no proof but, as Zinsser states, it seems reasonable to be content at present with the idea that as the result of antigen-cellular contact there is always represented in the antibody something of the antigen which endows it with its specificity.

The significance of antibody formation lies in its relation to the effort made to get rid of the invading antigen. Antibodies can be divided into two general classes, known as antitoxins and as sensitizers. Antitoxic immunity is not the rule and occurs only when there is produced a true soluble exotoxin or poison, the absorption of which is causing the disease. To the sensitizers, constituting by far the larger group of the antibodies, belong the bactericidal, bacteriolytic, precipitating, agglutinating, tropinizing (opsonins), hemolytic, anaphylactogenic antibodies. They constitute active forces in the resistance that the body offers to infectious agents.

It is now fairly well established that in normal serum which has known antibactericidal qualities, there is a thermostabile substance comparable to a sensitizing antibody and, though present in but a very small amount, it must precede in contact with the antigen, before the second nonspecific thermolabile element, known as alexin, or complement, can act upon the antigen. Alexin may act with its own lytic power or, more important, render the antigen more susceptible to phagocytosis by the leucocytes and certain fixed tissue cells. In fact, the solubility of the antigen seems to be the determining factor as to lysis or phagocytosis rather than specific antibodies.

Specific cellular response constitutes the basis for the known facts of immunity. Because of its nondiffusibility the antigen stimulates cellular response simply by contact with the cellular surface. To this contact it is a known fact that cells respond specifically. Just how we do not know. There is a reaction product within the cell called an antibody and if, due to continued stimulation from the antigen or from a process once started within the cells, the amount of this specific product becomes excessive, it is thrown out into the circulation as a free antibody. In some mysterious way the cell retains a latent reaction capacity to the original protein molecule, even though circulating antibodies have disappeared. Increased specific cellular irritability may remain purely a cellular function. Naturally immune individuals possess this cellular reaction capacity independent of antibody formation.

The rôle of the sensitizing antibodies by antigen-antibody contact is to render the antigen vulnerable to certain changes which lead to its elimination. The resulting effect upon the antigen may not always be due to specificity of the antibody, for the result is perhaps as often due to environmental conditions. In fact, the specificity of antibodies, except as they will affect the vulnerability of a certain antigen, is denied by some. No claim is to be made that the destruction of bacteria is entirely dependent upon phagocytosis, but the fact remains that the degree of phagocytosis and recovery from infection go hand in hand.

It is necessary to consider serum disease, anaphylaxis and hypersensitivity on more or less of a common basis. So much of this work is in the research stage and the literature is so voluminous that one can but mention a few outstanding instances. Vaughn's work demonstrated the poisonous nature of substances derived from proteins (bacterial and nonbacterial), products of incomplete digestion and known as split proteins. In 1907, Gay and Southard attempted to show that in horse serum there are two elements, first, one termed anaphylactin, which remains in the serum of a normal animal after injection and brings about a change in the cells so they react violently to the second or toxic element of the horse serum when the second dose is injected. The toxicity, however, of the second element depends upon the previous preparation of the animal's serum.

Bezredka has denied this contention that anaphylactin is derived from the antigen but claims that it is an antibody derived from and developed by the body cells. Friedberger tried to show that a poison anaphylatoxin was always the same and the result of antigen-antibody reaction. Probably the most consistent conception is that advanced by Jobling and Petersen, who demonstrated a poison analogous to anaphylatoxin and the split proteins by the action of fresh serum on a great variety of substances—that is, even with starches and agar as well as proteins. They proved to their satisfaction the presence of a ferment in normal serum, which is held in check by an antiferment, and that when serum is brought in contact with another serum or protein the antiferment will be withdrawn, and that this allows the normal serum to proceed with the digestion of its own serum proteins, some of which fractions are highly poisonous. These poisons produced in the early stages of digestion and producing the same symptoms as in anaphylactic shock, are the result of proteolysis and include the split proteins of Vaughn, the anaphylatoxin of Friedberger, the proteotoxins, the serotoxins of Jobling and Petersen. An anaphylaxis to nonprotein substances occurs only when these substances are chemically bound to blood serum or other proteins.

Zinsser conceives that foreign substances enter the body and the cellular structures to be disposed of in two different ways. The first for the cells' nourishment and secondly to be broken down into simpler substances and thus eliminated. Since nondiffusible substances cannot be absorbed, there is produced by the cells, substances or antibodies, the formation of which is a direct attribute of nondiffusible substances. This union of antibody and antigen on the cellular surface permits the active constituents of the cells to functionate, and substances are to be classified according to their chemical structure, their diffusibility and to the ease with which they produce antibodies.

In the anaphylactic state it is the most acceptable theory that with the introduction of the antigen, for the first time, there occurs a primary injury to the capillary endothelium that renders it specifically permeable, and that while no antibodies are to be demonstrated circulating in the body fluids there does occur the

production of antibodies and their storage within the fixed tissue cells. With the introduction of the same antigen for the second time, there remains the permeability of the capillary endothelium with the antibody located intracellularly so that the capacity for union of the antibody and antigen is markedly increased and in a location to facilitate a most intense cellular reaction.

Antibodies fixed to the cells hold a most important position. Circulating antibodies represent an excess formed in and by the cell but thrown off early by the process of sensitization. Precipitins and anaphylactins are thrown off early in the process of sensitization.

In asthma, hay fever and human hypersensitivity the search for circulating antibodies has been disappointing. The allergic individual reacts in an abnormal way, yet many of the fundamentals governing this reaction have been conclusively answered. The positive and negative phases are difficult to explain. Also the negative skin tests with positive clinical proof and vice versa.

Prausnitz and Koester transferred specific hypersensitiveness by injecting the serum of sensitized animals, claiming the response to the specific protein to be due to the presence, in the serum transferred, of a specific substance known as reagin. Grove and Coca claim to have demonstrated the constant presence of reagins in the blood of asthmatics and hay fever patients. While reagins resemble precipitins, according to Levine and Coca, the reagin content in the blood stream does not change during treatment. In fact, after the reaction has subsided and the ordinary antibodies slowly disappear these related substances of antibody nature remain constant.

The various humoral theories are entirely inadequate and, just as in infections, emphasize the importance of cellular activities. Circulating antibodies in immunity so change the antigen before cellular contact as to prevent reaction. In serum disease the reaction is to be based on quantitative rather than qualitative presence of antibodies.

If antibodies are a necessary factor and are not to be found in the circulating fluids, then their demonstration within the cells is to be established. The so-called Dale reaction does demonstrate

this to be a fact—that is, their presence is shown in tissues that have been freed from circulating blood. The fixed tissue antibodies, those intimately associated with cellular structure alone offer an explanation for certain types of reaction and hypersensitivity.

Hypersensitivity in man is probably acquired, the original sensitization having been shown to be possibly due to a prenatal influence, for not only the passive absorption of antibodies but also the active absorption of antigen through the placenta has been demonstrated.

IN CONCLUSION.

The primary resistance to infections and foreign proteins is measured by the cellular reaction capacity. Secondary factors are certain substances, antibodies, in the humoral element of the blood that are themselves the product of the primary cellular reaction capacity.

Cellular phagocytosis is the most effective weapon that the cell possesses in eliminating foreign protein material.

A primary avidity for phagocytosis is not only retained by the leucocytic cells of the blood but by certain fixed tissue and wandering tissue cells.

The reticulo-endothelial system probably includes the cellular elements responsible for the production of antibodies; it constitutes a hematopoietic system and embraces those fixed tissue cells and wandering connective tissue cells that have retained the greatest avidity for phagocytosis.

Specificity of cellular reaction is the basis of infection control and immunity. It seems probable that when the secret of specificity of cellular reaction is known, then will the major problems of immunity and infection be solved.

The so-called reticulo-endothelial system seems to offer the most fruitful field for the investigation of this problem, first as the source of the humoral substances and the cellular elements of the blood that take an active part in combating infection, and secondly, it possesses cells that seem to have powers of selectivity.

The secret of cell specificity and selectivity in production and activity forms the crux of the problem.

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X.

SOME INTIMATE STUDIES OF NASAL FUNCTION:
THEIR BEARING ON DIAGNOSIS AND
TREATMENT.*

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ST. LOUIS.

The following study was undertaken, not with the idea of contributing new discoveries to the subject of nasal physiology, but of scrutinizing the work of the physiologists of fifty years ago in the light of present clinical experience.

This jump of half a century is not a random one. The fact is that most of the fundamental work in nasal physiology was done at that time to the satisfaction of physiologists who then turned their attention to other things; little has been done since to modify their ideas.

An attempt has been made to evaluate the importance of the various processes from a clinical standpoint as an aid in determining the types of treatment which should prove the most rational and useful in the varying states of nasal pathology.

CILIARY ACTION.

Cilia have recently been rediscovered by laryngologists; so much has been written on this subject of late that only the mention of some of the more practical phases of ciliary action will be made here.

To my mind, cilia are by far the most potent factor in maintaining and restoring nasal health. Unless one has watched them in action, and estimated the amount of work they do and the tenacity and persistence with which they do it, their importance will be vastly underrated.

*Read by invitation at a special meeting of the Section on Otolaryngology, New York Academy of Medicine, October 13, 1931, as part of the program of the Clinical Congress of the American College of Surgeons.

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Ciliary action has been studied for centuries. It was recognized before the nature of epithelium itself by A. de Heide, in 1683, in the membranes of mollusks.

Cilia do not propel foreign bodies by direct contact with them, but their concerted wave action propels the viscous blanket of mucus in which the foreign particles are caught. This propulsion goes on at a terrific speed—many times the length of the cilium per second. According to Hilding,¹ the dragging effect on the layer of mucus is so great that the cilia about the middle of the nose can clean the pre-turbinal areas, where no cilia exist, by tugging back the mucous layer. Ciliary activity in the nose is greatest in protected areas; in the meatuses and on the lateral surfaces of the turbinate margins, although it exists everywhere on the mucosa.

Ciliary currents in the nose are conceded to travel backward over fairly well established routes to the pharynx and into the esophagus, never toward the nostril. Within the sinus the ultimate convergence of the ciliary pathways is toward and through the ostium. But these trails are by no means by the most direct route. An auxiliary opening made in a sinus, away from the ostium, has little if any effect on them. Mucous streams pass it by, and continue to emerge through the ostium.

It would seem therefore that antrum "windows" were desirable only when ciliary action has ceased, or when, as a result of ostium closure, sufficient secretion had accumulated to nullify ciliary streaming.

Cilia of the nasal mucosa survive more abuse than is ordinarily believed. Cold, drying and certain drugs slow or stop their action; in a microscope demonstration of mammalian cilia (rabbit) which lasted all morning, they have been observed to stop several times and resume activity when the saline solution in which they were bathed was replenished.

The rhythm of ciliary waves and its causation, important as they are, are as yet imperfectly understood. It can only be stated, in passing, that ciliary activity is autonomic with the cell, and that while sympathetic nervous influence upon ciliary wave motion has been suggested, it has as yet not been proven.

Not only are the cilia less delicate than is sometimes indicated, but there is no doubt that they can and frequently do regenerate with the regenerated membrane, following the radical exenteration of a sinus.

The cilia, acting in groups, go through a definite cycle of activity. There is an "effective stroke" in one direction, in harmony with the other cilia in the area, then there is a "recovery stroke" in the other direction, by which the cilium returns in a comparatively flaccid state to its original position. In the rabbit's nose, for example, this cycle is performed several times a second. The activity varies in different areas and at different times. Portions of the membrane appear to rest while others work.

Returning to the amount of work which cilia perform, it is difficult to express this accurately, as the methods of measurement thus far have depended upon the movement of foreign bodies upon their surface, which introduces variables and inaccuracies. The classical figures of Bowditch,² based upon the propulsion of particles of known weight up inclined planes indicate a lifting power of 7 gram millimeters per minute, per square centimeter. These cold figures do not quickly convey the magnitude of the effort.

It may clarify the situation to put it thus: If each cilium were magnified to one foot in length, a correspondingly large antrum would be a mile high and have a capacity of 735 billion cubic feet—which is equivalent to the water supply of New York City for fourteen years and five months!—and yet this mat of one-foot cilia would clear it in a few hours.

What one gathers from such a performance is that a bacterium settling upon a membrane thus protected will not remain long enough in one place to divide and multiply. It is easy to see why normal sinuses are sterile. To begin with, air circulation through an ostium is infinitesimal, as will be shown, and the likelihood of bacteria entering the sinus through it in fairly large numbers is remote. Let this vigorous streaming be stopped, however, through toxins, drugs, drying or cold, or let the ostium become closed, so that the mucous coat becomes too deep for the cilia, then counter currents are set up by gravity, stagnation occurs and bacterial growth proceeds.

Cinemicrographs of ciliary action in rabbits were shown with this presentation.

Inasmuch as they are the front line of fortifications, it should be of utmost importance to us in planning the management of a case to know whether or not the cilia are functioning, or can be made to function.

Let us examine critically some of the other forces involved in nasal physiology: posture, air circulation in nose and in sinuses, respiratory pressures and fluctuations, humidification and radiation. It will presently appear that in order to apply our findings practically each of these factors must be regarded from three distinct and vitally important angles: First, from that of a normal nose; second, of a recently normal nose, temporarily deranged and capable of restoration to complete normality; third, from the angle of a nose altered by the ravages of disease and incapable of complete restoration by any means. We will return to this classification in summarizing.

POSTURE.

A study of sinuses in animals leads one to the conclusion that posture can have little if any effect upon drainage so long as the mucosa is healthy. The mucus which coats such a membrane is spread in an exceedingly thin, tenacious film on the surface of the rapidly moving ciliary mat, as we have seen. In such a system gravity has little effect compared to surface tension and ciliary action.

Apparently it is only after this mechanism is somehow upset in such a way as to increase the bulk of surface mucus that gravity enters into the equation. This may be due either to an increase in production of secretion or to an obstruction of the ostium preventing its escape.

The increased film of moisture is beyond the effect of ciliary action; it accumulates at the bottom of the sinus cavity. If the inflammation advances, change of posture can aid drainage only for a short time; that is, only so long as the ostium remains open or there is an artificial opening. Posture, therefore, so far as spontaneous drainage is concerned, plays an exceedingly limited part.

AIR CURRENTS.

If one would study the literature dealing with the pathways of air currents in the nose, he must turn back, as stated, to the '80's and '90's. In those decades a dozen American, English, French and German authors reported their findings and very little has been said about it since. According to Goodale, Lambert Lack and others,³ the stream of inspired air does not pursue a straight course from nostril to choana, but passes, in the main, in a wide curve beginning at the nostril, extending through the olfactory fissure and ending in the upper part of the choana, thus practically avoiding the inferior meatus entirely, except for small eddies which curl against (1) the face of the sphenoid; (2) down

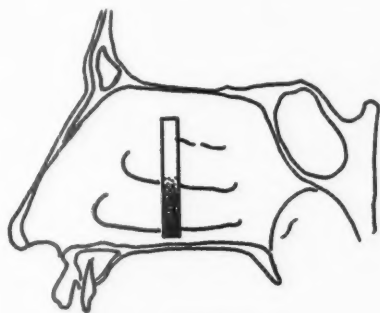


Fig. 1. Inspiration.

over the inferior turbinate, and (3) down in the vestibule. "Hence," says Lack, "nasal stenosis may be complained of if the middle meatus is blocked, even when there is a free airway below this level."

Our experiments agree with this in every way. They do not confirm, however, his second statement that "expired air travels chiefly along the inferior meatus; hence the difficulty of blowing secretion from the nose until it has reached the floor of the cavity."

Our conclusions are based upon two entirely independent groups of experiments. The first was crude but convincing. Small squares and strips of blue litmus paper were moistened

with distilled water and placed in various locations in the nose. The subject was then allowed to inhale the fumes of hydrochloric acid and the color changes of the litmus were observed.

The following precautions were observed to guard against errors due to diffusion: The subject was blindfolded and his attention was not directed to his respiration in order to avoid any irregularities due to his attempts at co-operation. The bottle was held to his nose for only a fraction of a second in the middle of an inspiration, quickly removed while inspired air was still passing to clear the nasal passages of fumes, and the nose closed so that the breath was expired through the mouth. An interval of several respirations was allowed to elapse and the performance

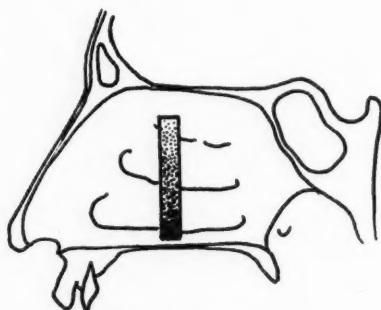


Fig. 2. Expiration.

was repeated. After about five such inspirations, the papers lying in or opposite the middle meatus and above that point were invariably red, those below it invariably blue. A long narrow strip of paper plastered against the septum midway back, and extending from top to bottom, came out particolored, the dividing line being approximately the lower edge of the middle turbinate. Acid fumes gently pumped into the mouth during expiration so as to obtain expiratory effects without jeopardizing the lung, gave much the same results with the litmus paper. The top turned red, the bottom remained blue—the difference being that the line of demarcation was somewhat lower and much less definite.

The second group of experiments was of a different sort, but the results corresponded in every respect with those of the first.

A head was sawed sagittally, exactly through the septum, and again in parallel planes, cutting the outer walls of the ethmoidal and maxillary sinuses respectively. These sections were clamped between plates of glass so that the nasal chamber could be observed on one side (the glass occupying the position of the septum), and the sinus cavities on the other (the glass on that side replacing the lateral wall of ethmoid or maxillary sinus, depending upon the section used). A rubber tube was fitted into the trachea, and wet cotton was packed between the plates surrounding the entire preparation, making it air tight but leaving the nostril accessible. Artificial respiratory currents were now set up through the

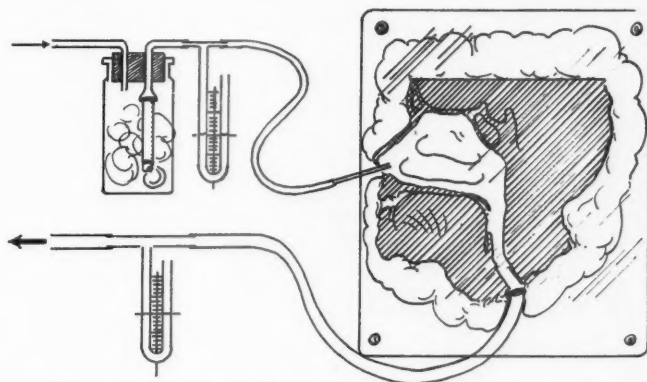


Fig. 3. Method of studying nasal air currents in the cadaver.

tube in the trachea, the pressures being maintained as nearly normal as possible, by means of manometers in the system. While these currents were passing, thin streams of smoke were introduced through small cannulae into various portions of the nasal chamber and their pathways studied. By this means, a steady inspiration or expiration could be maintained for an indefinite time and the smoke currents carefully controlled and photographed. The normal fluctuations could also be reproduced.

For photographic purposes, it was found advantageous sometimes to fill the nose first with smoke, and to observe the current of clear air passing through it, as these were established by the

breathing apparatus. (Both the smoke trails in air and air trails in smoke are recorded as a motion picture.)

To study diffusion, small pieces of blotting paper moistened with hydrochloric acid were placed in the sinuses. By employing ammonia fumes in the respiration apparatus, the white clouds of ammonium chloride could be made to serve as indicators of the behavior of the air about the ostium. To our surprise, after twenty respiratory cycles, there was not enough of a chloride cloud to record with the camera. The same may be said of the smoke experiment. After half an hour's work with the air currents in the nose the sinus contents were rarely even hazy.

The results of these smoke experiments correspond to those of Lack, adding beside, a distinct inspiratory eddy in the naso-



Fig. 4. Expiratory currents.

frontal angle. It was quite apparent from watching the trails that the direction of current was determined by the shape and position of the inlet (nostril) and the angle at which the inspired air impinged against the slope of the bridge of the nose. The choana, being larger than the nostril did not modify, by restricting it, the flow and shape of the current as it entered.

On expiration, however, conditions were reversed. Now, the air entered the choana uniformly throughout its extent, again passing upward, influenced by the angle of the face of the sphenoid, describing much the same curve as it did on entering, except that the channel of greatest velocity was much broader. The rel-

ative sluggishness was caused by the retarding action of the restricted exit (nostril), and the current became accelerated in a narrow channel only as it prepared to leave the nose.

The inferior meatus, indeed, conducted a definite channel of air only when excessive pressure was brought to bear, either by forced respiration or by restricting the nostril. There was a distinct triangular "air pocket" or dead calm area on the floor of the nose posteriorly. According to our findings, therefore, the difficulty of blowing secretion from the nose while it is still in the middle or superior meatus, is due rather to the disseminated and dispersed nature of the outgoing blast than to the current flowing through the inferior meatus.

It appeared also that employing a head with thin and shrunken middle turbinates or one with an amputated middle turbinate did not materially alter either the inspiratory or expiratory currents.

Introducing obstacles of any description either on the septal or the lateral wall produced the most pronounced changes, in that the entire current was broken into eddies and whirls.

On inspiration, no current could be made out to pass beneath the middle turbinate. On expiration, however, a distinct whirl could be seen to emerge from beneath it, which is very definitely recorded in the motion picture.

AIR PRESSURES.

In 1877, Braune and Clasen⁴ measured the pressures occasioned in the maxillary antrum by the fluctuations in normal respiration and in forced inspiration and expiration. They found these ranging from -6 to $+6$ mm. of water with normal respiration and from -50 to $+35$ with forced efforts.

In 1922, Rockwell Coffin⁵ stated that he found a positive antrum pressure averaging 1.17 cm. in apparently normal noses, with the respiration at rest.

Regarding the situation theoretically, it is difficult to see how, with an open ostium, there could be more than the minutest difference between the pressure in the sinus on one side of the ostium and the nasal chamber on the other, and this only during respiratory air flow. With breathing at rest, nose and sinus must both be at atmospheric pressure. It is conceivable that after a time, gases emitted from or absorbed by the mucosa (if this occurs),

might create a plus or a minus pressure, provided the ostium were tightly closed.

Twin water manometers were accordingly prepared and connected with two similar antrum puncture needles. One of the needles was introduced into the antrum via the inferior meatus. the other was held with its tip midway back in the nasal fossa.

First of all, the figures of Braune and Clasen were verified. The antrum manometer registered, in relatively unobstructed noses, from 4 to 7 mm. of water pressure plus and minus, averag-

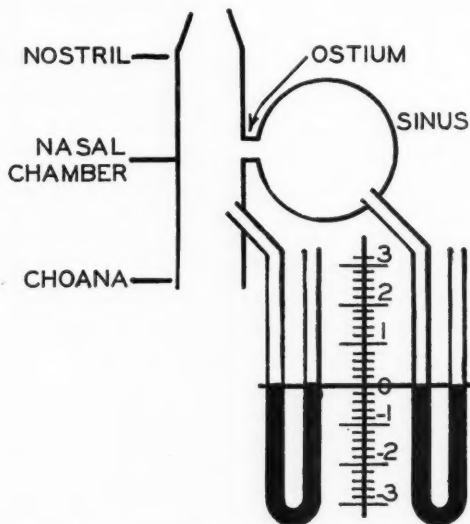


Fig. 5. Scheme of manometers in the nasal chamber and the sinus.

ing a total fluctuation of 10 mm. As was to be expected, the pressures in the nasal chamber corresponded closely to those in the sinus so long as the ostium remained open.

A glass and rubber model was now constructed, by means of which the patency of openings representing the ostium, the nostril and the choana could be controlled. The twin manometers were adapted to these. With this model it was possible to duplicate the observations made in the nose, which were as follows:

1. So long as the ostium remains at all patent, the pressure changes in the sinus are identical with the pressure in the nose.
2. As the ostium narrows, a progressively increasing time-lag occurs in the fluctuations of pressure within the sinus, so that it requires an appreciable time for the equalization of pressure on the two sides of the ostium to occur.
3. Regardless of this time-lag, the peak pressures are not reduced until the ostium finally closes altogether, when they cease entirely.
4. On restriction of the nostril alone, the peak pressures, both plus and minus for a given respiration, were increased in both the nasal fossa and the sinus.
5. On restriction of the choana alone, the peak pressures, both plus and minus for the same respiration, were reduced in both the nasal fossa and the sinus.
6. On restricting both nostril and choana, the pressures varied between wide extremes in direct proportion to the relative restrictions.

The contemplation of these extremely simple physical phenomena may seem a bit futile, but immediately a highly practical application presents itself, which may be simply stated thus: Variations in the speed or promptness of sinus fluctuations represent changes at the ostium; variations in pressure peaks represent conditions in the nasal chamber. Concretely, it operates thus: Diagnostic puncture has been performed. Immediately after the needle is inserted and before any other manipulation is done, a manometer is attached for a moment.

Fluctuation: ostium open.

No fluctuation; ostium shut.

Fluctuation only on forced respiration; ostium swollen shut but still penetrable.

Fluctuation slow to reach peak: ostium narrowed.

Reduced excursion of fluid on normal breathing (diminished peak pressure): nasal obstruction behind the ostium.

Exaggerated excursion on normal breathing (increased peak pressure): nasal obstruction anterior to the ostium.

If the membrane is swollen and the ostium is obstructed, the pulse beats may be observed in the manometer.

This is a fair amount of important information for so simple a procedure. The manometer is quickly made from a piece of glass tubing bent "U" shaped and half filled with water. It need not be calibrated. The behavior of the fluid is definite and surprisingly characteristic. The tube should be attached to the needle before puncturing or, if attached afterward, the connection should be by means of a bayonet joint and not by slipping the rubber hose over a glass adapter, as this creates a positive pressure in the system if the ostium happens to be closed.

In no case was it possible to demonstrate the positive pressure described by Coffin⁵ with respiration at rest, so long as the ostium was open—that is, a case in which the normal fluctuations occurred during respiration.

What is the importance of these pressure changes in the nose? Why should they occupy our attention? Let us analyze for a moment what the manometer reveals.

An atmosphere of pressure regarded at sea level as 760 mm. of mercury is equivalent to a column of 10,260 mm. of water. The antrum fluctuation of 10 mm. then represents $1/1026$ of an atmosphere pressure. According to Boyle's law, therefore, it expands and contracts $1/1026$ of its volume with each respiratory cycle. This means that in an average healthy sinus of 15 cc. capacity the air exchange with each breath is about 14 cmm. In an ethmoid cell of 1 cc. at the same time the exchange must be a little less than a single cubic millimeter.

What can this accomplish? Is it of enough importance to engage our further attention? It could scarcely be regarded as a draught through the ostium, although it probably suffices to keep the latter free of films and bubbles of mucus which may interfere with ciliary drainage.

If each expiration introduced this much fresh air and each expiration extruded an equal amount of stale air, it would take almost an hour to effect a complete change. But this cannot be the case. In all likelihood most of the fresh air injected at each expiration (warm, moist air from the lungs) is ejected again at the next inspiration so that the exchange must be infinitely slower and must depend largely upon diffusion. The limited extent of diffusion has already been commented upon.

HUMIDIFICATION—RADIATION OF HEAT.

Humidification and warming of inspired air, phenomena which we accept without much thought, are accomplished with amazing efficiency by the nose.

The tidal air which passes through the nose in twenty-four hours approximates 500 cubic feet—the contents of a cubical box eight feet high. During the twelve or so hours of inspiration, our noses saturate these 500 cubic feet and raise their temperature from whatever it may have been to nearly 98° before it reaches the pharynx.

In accomplishing the former function it evaporates over a litre of water. The calories of heat expended during this time in warming the air from an initial temperature of 20° C. to body heat were estimated by Helmholtz⁶ at 70,000 small calories.

These two facts alone should be of enormous importance in planning the mechanical phases of operations on the nose. Four turbinate bodies perform the major part of humidification and warming. The pharynx, larynx and trachea are unequipped for these functions. Suppose we find it necessary to remove a turbinate; two things result. Not only are the lungs deprived of a proportional part of the warmth and moisture which this turbinate supplies, but some area just behind it is deprived of practically all of its warmth and moisture, for which it is in no way equipped to compensate. If this happens to be a lip of the eustachian tube or a sphenopalatine ganglion, the irritation may produce immediate effects. If it is a less specialized area, the drying and cooling of the cilia soon interfere with their action and deprive the area of its chief protection.

Many patients predict the advent of a "cold" when they experience a burning sensation in the pharynx or on the back of the velum palati. We know that when this happens, the cold has already begun. Some localized swelling has occurred, forcing all the inspired air to enter through a narrow channel by no means adequate to warm and moisten it. Its projection against some point in the pharynx produces the "hot spot" complained of, which at first is a mere by-product of the cold, but which soon becomes intensely irritated, accumulates secretions, and adds to the conflagration. Many a cold may be aborted if the nasal circulation

can be re-established early, before violent irritation occurs. I fully believe these spots to be largely responsible for many of the postnasal colds which travel down to eventuate in a laryngitis or a tracheitis, the process traveling by the lymphatics beneath the surface and not upon it.

If we go a step farther and remove at the same time the entire face of the sphenoid sinus, we introduce the cold dry air into it. Thus we destroy at once two temperature buffers: the radiator-like turbinate and the sphenoid face. The ill effects which may follow such an operation are not theoretical; furthermore, the task of restoring function in such a nose is hopeless. It should not be inferred from this that I am condemning the removal of turbinates and the drainage of sphenoids, or that I do not perform them in selected cases, but I do submit that the facts presented should be thrown into the balance before operating, and exploratory or experimental operations involving extensive removal of tissue should be resorted to only when grave and pressing indications exist. I have seen the severest headaches relieved by a wide opening of the sphenoid, following removal of a turbinate. These operations were abundantly justified, by the relief of headache, even though the patient's nose could hardly be regarded as a normally functioning organ afterward.

Two things puzzle one: First, if the nose normally gives off a litre of water per day, what becomes of it in cases of choanal atresia, say a unilateral one? Can it be that the secretory mechanism is so regulated or so precisely stimulated by the passing air that it is possible for one side of the nose to throw off its 500 cc. of fluid while the other suppresses it entirely?

Second, most mucus secretions contain about 5 per cent solids. What becomes of these? Do they constitute the tenacious layer which floats upon the cilia to be ultimately swallowed? It seems likely. Furthermore, what part do they play in the unevenly ventilated noses which produce the dry spots already referred to?

The idea that the sinuses aid in warming and humidifying inspired air is difficult to comprehend. Certainly the $\frac{1}{1026}$ of the sinus content, mentioned as passing with each respiration, could have little effect on the inspired 500 cc.; besides half of it enters during expiration from the lung and is already warm.

But these air jackets of the nose undoubtedly act as admirable temperature buffers between the cold air and the rapidly chilling turbinates on the one hand and the delicate nervous structures which adjoin them on the other. The minute quantities of air which pass into the sinus with each respiration may modify the temperature within—how much one is unable to say.

SUMMARY AND EVALUATION.

What conclusions may be drawn from these limited observations which will be of definite clinical value? One is convinced that ciliary function is the greatest physiologic factor in preventing and restricting infection, and that wherever it exists the first consideration should be to maintain and further it. Other functions are important directly as they contribute to it.

Where no ciliary help is to be expected, the more obvious mechanical means must be resorted to, to fit the individual case.

Always mindful of the fact that it does not include cytology and immunology, the following evaluation suggests itself:

I & II

1. CILIARY ACTION + + +
2. AIR DISTRIBUTION +
3. HUMIDIFICATION +
4. DIFFUSION
5. RADIATION
6. AIR PRESSURES -
7. POSTURE -

III

1. CILIARY ACTION + +
(IF ANY)
2. AIR DISTRIBUTION + +
3. HUMIDIFICATION + +
4. POSTURE
5. RADIATION
6. DIFFUSION
7. PRESSURES

Fig. 6. I and II, evaluation of factors for normal noses and those capable of restoration to normal; III, for chronically inflamed noses.

No new facts have been brought to light in the course of this study. If there is any justification for again holding up to scrutiny the intimate workings of the nose in its homely functions, it lies in the embarrassing realization that every discovery that was ever made has been spread before our unseeing eyes since the beginning, and that if new knowledge is to be had of the nose it behooves us first to be fully and surely conversant with what has gone before.

1010 BEAUMONT BUILDING.

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XI.

AN IMPROVED METHOD OF COLLECTING NASAL CULTURES.*

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ST. LOUIS.

The method described here is particularly adapted to collecting cultures for special investigations where it is important to exclude contamination from the external nares. Many pieces of research have been based upon results secured by simply passing a cotton swab through a nasal speculum, trusting to luck and a steady hand to prevent as much contamination as possible.

Various methods have been described in the past, such as sterilization of the vestibule with mercuric chloride, Harrington solution, etc., or the sterilization of a swab in a rubber cot from which it is later projected. None, however, have proven very satisfactory, due to ineffectiveness or labor involved.

The outer glass sleeve is about 4 cm. long and 7 mm. outside diameter. It is flared on the end by means of a fine gas flame and a carbon, such as that used for balopticans, and which has been sharpened like a lead pencil. The maximum diameter should not exceed 1 cm., since the average nares will not accommodate a larger tube. The inner sleeve is prepared in a similar manner from 4 mm. glass tubing cut 6 cm. long. The flare on this tube is made with a finer pointed carbon and adjusted to telescope with the outer tube in such a way as to leave the outer tube projecting a short distance beyond it. The end of a wooden applicator is dipped in collodion and a small tightly wound swab put on.

The swab must be small enough to fit snugly within the flare of the inner tube. Before adjusting into place, the swab is passed rapidly through a flame to remove the straggling fibers

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Single sleeve arrangement.



In telescoped position for insertion into the nares.



Extended position for swabbing mucous membrane.

of cotton. The apparatus is assembled, placed in a 6-inch test tube, sterilized and stored ready for use.

Various modifications of this method may be used. The cultures are easier to take, but sterility is less certain if a single sleeve tube is used. There is less assurance of protection from hairs and straggling cotton fibers. However, in infants where the nares are small, the large size tube will not enter. It may be necessary in such instances to use the inner tube alone with the edges rolled outward slightly to protect the swab as much as possible. If a larger tube is used a constriction should be made just back of the bell-shaped flare in order to hold the swab in the center of the tube. The spread between the inside diameter of the inner tube and the outside diameter of the outer tube may be reduced by using thinner material, such as metal, for making the sleeves.

Experiments show that results secured when using this apparatus are quite different from those secured when using the nasal speculum alone.

Cultures are taken by inserting the whole apparatus into the nares through a nasal speculum.

XII.

THE SIGNIFICANCE OF ALLERGIC NASAL AND SINUS DISEASE IN RELATION TO ASTHMA.*

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For many years it has been noted that a very large percentage of asthmatics suffer from nasal and sinus disease. In many the nasal manifestations have been limited to comparatively simple conditions and complaints, such as mucoserous discharge with sneezing, and occasional obstruction from temporary edema. In others the nasal changes have taken extreme forms, the commonest being chronic nasal blockage from extensive edematous swelling of the mucosa with very free mucoid or mucopurulent secretion. In addition, masses of edematous membrane often fill the sinuses; or very frequently the grosser edematoses, beginning in the sinuses, usually the antra and ethmoids, may extrude through the ostia and appear in the nose as polypi. Also, polypoid degenerations of the middle turbinates may develop, and upon all these conditions may be superimposed more or less extensive suppuration in the sinuses, resulting from lowered tissue resistance, mechanical interference with drainage, and secondary invasion of infecting organisms.

In nearly all such cases the nasal and sinus membranes will be found to manifest well defined histopathologic characteristics. These consist primarily of edematous infiltration of the subepithelial stroma of the mucous membrane, the epithelium showing striking evidence of hyper-secretion with desquamation. There is also an accompanying cellular infiltration in the tunica propria made up largely of the highly characteristic eosinophils, in addition to the normally present lymphocytes and plasma cells. The serous swelling of the subepithelial stroma results in epithelial hyperplasia, and in the more chronic cases fibrous tissue hyper-

*Read before the Western Section of the American Laryn., Rhino, and Oto. Soc., Los Angeles, January 29 and 30, 1932.

plasia of varying degrees. Occasionally a retention cyst is formed from blockage of the outlet of a mucoserous gland. Small sub-epithelial abscesses may rarely be found, and in addition bacterial invasion of the mucosa in the form of organisms in the tunica propria has been reported by Kistner¹ in his paper on nonpurulent hyperplastic sinusitis. Pseudocysts are frequent, and consist of large serous collections in the distended tissue spaces beneath the epithelium.

These findings have resulted in much speculation regarding the relationship of nasal and sinus disease to asthma, with the very common conclusion that asthma is caused by nasal and sinus disease, and that it may be cured by surgery directed to the eradication of troubles in the upper respiratory tract. Consequently, much effort has been expended by rhinologists in futile attempts to cure asthma by means of complete extirpation of diseased sinus mucosa. The industrious surgeon has had his failures deplored by his more industrious brother, with the assertion that he has not been radical enough—he has not removed all of the diseased tissue from the sinuses. Hence the sinus holocaust, with the sacrifice of countless innocent lining membranes whose only offense was that of being the seat of an allergic edema.

Several years ago I advocated the theory that the sinus disease which so frequently accompanies asthma is simply the manifestation in the sinuses of the same phenomenon which, when occurring lower down in the respiratory tract, causes asthma—that the two are associated manifestations taking place in membranes of almost identical histologic characteristics and are expressions of the same underlying condition and neither one the cause of the other. I made then and make now no claim for priority in this theory, as it has been held by many others besides myself. It excited little comment at the time except to brand its advocates as skeptics and ultraconservatives. The theory was a difficult one to prove, and in support of their contention its opponents had the indisputable fact that asthmatics often do improve after sinus operations and that studies of sinus lymphatic drainage, such as those of Mullin and Ryder,² seem to point to the possibility of direct association between sinus disease and bronchial conditions. However, time has passed, and the

opponents of the theory have proceeded to prove it for its advocates. Sinus operations for the cure of asthma have been performed by thousands; radical and conservative, by good and bad alike, and still the asthmatics continue to wheeze. Not only does the asthma persist, but the sinuses themselves are not cured. Polypi recur, excessive secretions continue to form, and patients return to the rhinologist for treatment and operation, operation and treatment, until finally the mere mention of the word sinus produces in the average person a state of nervous apprehension second only to the dread excited by the most serious and fatal diseases.

Still we have with us staunch advocates of this form of radical surgery, citing impressive statistics of patients cured, but in most instances neglecting to tell us how long they have remained cured. With one often repeated statement of these enthusiasts, I fully agree. Most of us have not removed all of the diseased membrane from the sinuses of our asthmatics. To do so would often be a stupendous undertaking, necessitating frequently the opening of the farthest reaches of every accessory cavity and the removal of every vestige of mucous membrane contained therein. It is an undertaking from which I shrink with fear, and I believe that in most cases it is impossible of accomplishment. But even if that were not true, and if it were possible to remove every vestige of diseased mucous membrane from the sinuses, still we have not removed the disease itself. It resides in less surgically accessible portions of the body, because it is fundamental in the individual and consists in the "capacity to develop hypersensitivity to foreign substances." In short, it is allergy, and the nasal and sinus disease which usually accompanies asthma is merely a manifestation of allergic disease in the nasal and sinus membranes, just as asthma is a manifestation of allergic disease in the bronchial mucosa. The two are associated conditions, both dependent upon an underlying state of hypersensitiveness. Neither bears etiologic relationship to the other.

Here should be added, for the sake of further emphasis and for comparison, a description of the histopathologic picture of the bronchial mucosa in asthma. The pathology is strikingly similar to that found in the membranes of sinuses which are the

seat of allergic disease. The most constant change is an edema of the bronchial mucosa, a serous infiltration of the subepithelial connective tissue, with marked eosinophilic infiltration. The eosinophils are accompanied by small lymphocytes and plasma cells, and some connective tissue hyperplasia is present, both around the arterioles and in the tunica propria. Hypertrophy of the smooth muscle of the bronchial walls is conspicuous by its absence in many cases, raising the question whether bronchospasm is ever really an important characteristic of the asthmatic attack. Bronchoscopic observation has led me to the belief that the attack is caused primarily by an edematous swelling of the bronchial mucosa and concomitant accumulations of mucus, and not by spasm of the bronchial musculature. In this connection, it is only fair to state that smooth muscle hypertrophy in the bronchial walls in asthmatics has been observed by Fisher and Beck,³ Murphy and Case,⁴ Harkavy,⁵ and Huber and Koessler,⁶ as recorded in their reports on the pathologic findings in fatal cases of asthma. On the other hand, Steinberg's work,⁷ based on careful experimental, clinical and postmortem observations, bears out my own opinion.

In any event, comparison of histologic findings in the bronchial and sinus mucosa of asthmatics will impress the most superficial observer with the close similarity of the pathologic changes manifested in both, adding a further powerful argument in support of the contention of the paper. An additional observation of importance in this connection is made by comparison of the nasal and bronchial secretions from cases of bronchial asthma with associated nasal disease. Here, in the uninfected cases, we are struck by the similarity of the microscopic picture. Eosinophils are present in large numbers in the secretion from the bronchi as well as from the nose, and even in the secondarily infected cases numerous eosinophils will be found in company with the polymorphonuclear neutrophils which may then predominate.

It may be of value to quote certain statistics which have been derived from a study of my own practice. Out of 2000 case records analyzed with the idea of discovering the incidence of allergy in rhinologic practice. I have found that 700 were either primarily nasal complaints or were sent for the purpose of nasal

examination for possible focal infection or other relation of nasal and sinus disease to remote pathologic conditions. Of this 700 cases, 191, or approximately $27\frac{1}{3}$ per cent, were allergic. Of this 191 allergic cases, 57 were found to be associated with asthma, bronchitis and bronchiectasis. Of these 57 cases, X-ray demonstrated the presence of sinus pathology in 48. The X-ray was negative in two, and seven were not studied radiographically. Therefore, of the 50 asthmatic and bronchial cases studied radiographically, 48, or 96 per cent, were found to be suffering from some form of sinus involvement. Of these, six were the seat of a sinus suppuration. The other 42 were of the so-called hyperplastic type so characteristic of allergic disease in the sinuses. In 19 of the 57 cases surgical operations were performed on the nose or sinuses, or both, but without permanent beneficial effect on the chest condition in any case. This is not a large enough series to be of great value in proving the main point of the paper, but it bears out the experience of many others, both internists and rhinologists.

If we admit that allergic nasal and sinus disease is not a cause of asthma, it is important that the true relation each bears to the other should be defined. That relation is exceedingly simple. Nasal and sinus allergies are forerunners of asthma; they are finger posts pointing the way, and they tell us plainly that the patient is very likely to develop asthmatic trouble farther along the road. If the rhinologist will think a moment he will realize that of the many cases sent to him for examination of the sinuses in asthma, nearly all will give a history of some form of nasal trouble existing antecedent to the asthma. This is not always true, of course, and some asthmatics are not conscious of having had nasal trouble at any time, but most of them have had it, and careful questioning will usually elicit a very definite history. Many have not been sufficiently handicapped by the nasal manifestations of their allergy to seek relief, or the condition has been seasonal or occasional, and hence has been neglected, but in a very large majority of cases nasal allergy in some form has preceded the allergic manifestations in the bronchi, frequently by many years.

It is important, then, that nasal manifestations of allergy should be recognized as such and properly treated, regardless of the

slight degree of disability which they may be causing the patient, as it can be safely predicted that if untreated a very large percentage of such cases will later develop more serious and disabling conditions lower down in the respiratory tract. Once the early allergic patient is convinced of the nature of his ailment and educated to watch for manifestations of his allergy, he is very likely to be enabled to carry on a normally active existence entirely free from manifestations of his disease, even though he remains potentially susceptible to sensitization reactions throughout his life. In following this suggestion and pursuing the treatment of nasal allergy, it should be kept very prominently in mind that the removal of the manifestations of the allergic disease by mechanical means is not the cure of the disease. It is no more logical to expect to cure the allergy by removal of the edematous membrane covering a turbinate or lining an accessory sinus than it is to expect to cure urticaria by excision of the wheal.

It is true, of course, that other nasal and sinus conditions, such as infections, the presence of malignant growths, and even mechanical irritation, cause edematous changes in the nasal and paranasal mucosa, but space does not permit an exhaustive consideration of the subject of differential diagnosis of allergic disease. However, the vast majority of nasal edemas in asthmatics are allergic, and can be so diagnosed. When such is the case the present state of our knowledge requires that we discover the causes of the individual's sensitization and administer treatment calculated to remove those causes or to desensitize him. How this shall be undertaken is another matter and one not intended to be covered in the present paper.

Before final conclusions are drawn, it should be emphasized that I do not intend to ignore the fact that allergic nasal and sinus disease predisposes to suppuration; and, therefore, that an asthmatic may and often does have suppurative sinus disease along with his asthma. The paper should not be construed as an argument against needed nasal and sinus surgery in asthmatic patients.

On the other hand, based on what seems to have been ample observation, it has been my purpose to lead up to the following conclusions:

1. That nasal and sinus disease is primarily not an etiologic factor in the causation of asthma.

2. That asthma and nasal and sinus disease are often concomitant manifestations of the same condition in two different localities in the same patient, both dependent upon the same underlying etiologic factors.

3. That nasal and sinus operations should not be performed primarily for the cure of asthma, only such surgery being done as would be indicated by the same findings in the upper respiratory tract of the nonasthmatic patient.

4. That allergic manifestations in the nose and sinuses should serve as warning finger posts, pointing the way to danger, and that the patient should be so advised and should have his state of hypersensitization studied and treated—not only as a therapeutic measure directed to the cure of nasal symptoms but as a prophylactic measure calculated to prevent more serious consequences later.

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XIII.

BILATERAL DIFFUSE SUPPURATIVE LABYRINTH- ITIS, WITH DIFFUSE SUPPURATIVE LEPTO- MENINGITIS, COMPLICATING AN APPAR- ENTLY UNILATERAL OTITIS MEDIA.*

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CLINICAL REPORT—GUGGENHEIM.

This unusual case presents a "normal" ear more diseased than the known diseased ear. Although a complete diagnosis would in no way have prevented death from meningitis, which was already present at the first examination, it would nevertheless have been a satisfaction to those attending the patient had it been made. Postmortem microscopic examination revealed pus and a heavy submucosal infiltration, not only in the obviously affected but also in the clinically normal tympanic cavity. The labyrinth of the latter side showed even greater destruction than the other. Failure to do a caloric test on the left side allowed that ear to remain unsuspected until sectioned. It is unfortunate that after careful sectioning and studying of both temporal bones no certain pathway of infection could be found from the original focus (right tympanum) to the meninges and finally to the opposite (left) ear. This was due partly to an imperfect removal of the temporal bones at autopsy.

From certain sections one would be justified in saying that infection had entered the right cisterna in the region of the oval window, were it not for the fact that a fracture in this region, at autopsy, permitted pus to spill into the labyrinth. As to the involvement of the "normal" ear, the reasonable assumption is that infection traveled from the leptomeninges through the perineural and perivascular spaces of the internal auditory meatus to the labyrinth, for pus was found in these areas, more in the

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internal auditory meatus than in the labyrinth, and more in the labyrinth than in the tympanum. As stated, clinically the left drum membrane never at any time showed the least deviation from normal; nevertheless, the tympanic mucosa showed infection. Both labyrinths showed a diffuse suppuration, but, strangely, the side with the manifest otitis media showed far less destruction than the other.

We have learned from this case, among other things, the importance of a caloric test of both ears in every case of suspected internal ear or intracranial disease, and the fact that an infection of tympanic mucosa can exist with a perfectly normal drum membrane (this excludes the lack luster, the dirty, gray membrane, etc.).

CASE RECORD.

M. L. H., female, age 13 months, was brought to the Out-Patient Department of the Jewish Hospital on May 13, 1930. Temperature 102 R. Examination revealed a reddened drum membrane, right; left negative. Neck rigidity present. Chest and abdomen negative. Very rapid pulse. Kernig negative. The following history was obtained: Family record, no bearing; normal birth, normal development, normal appetite; sat up at proper time and prior to present illness was walking a little. No vaccination. No previous illness of any kind.

Present illness: Child was well until May 11th, at which time it suddenly became feverish and vomited. On May 12th the family physician was called and incised the right drum membrane. There followed a sero-sanguineous discharge. From the onset the baby had been drowsy.

Physical examination: Well developed and well nourished girl of 13 months, lying quietly in bed but showing irritability. Neck rigidity not marked but passive movement of head painful. Skin warm and moist. No eruption. Color fairly good. Temperature 102 R. Anterior fontanelle open, soft, not bulging. No photophobia. Ears: right drum membrane red and covered with a dry sanguineous exudate; left drum membrane negative. Mastoids: no redness or swelling but tenderness over right. Mouth negative. Fauces negative. Pharynx slightly reddened. Nose: negative. Heart and lungs negative. Abdomen negative. Extremities: free motion, negative.

Neurological: Kernig and Brudzinski negative. Reflexes not hyperactive.

Impression: Meningismus or meningitis complicating O. M. P. A. Dextra.

May 13, 1930: Cisterna magna puncture, 5 c.c. clear fluid. Operation upon mastoid not permitted.

May 14, 1930: Patient pale and apathetic. No conjugate deviation of eyes. No deviation of head. No nystagmus except for one rotary jerk to right, looking forward. Definite neck rigidity. Kernig negative. Pupils

equal and of normal size; both react very sluggishly to light. Throat and nose negative. Drum membrane: right, whitish and bulging; left, negative. Shrieking cries awake and asleep.

Operation: Myringotomy right followed by immediate flow of sero-sanguineous fluid. Culture made. Permission for mastoidectomy not obtained.

Radiograms of mastoids: The density is definitely increased in right as compared to left; however, no definite destruction of trabeculae on right. X-ray of chest: hilus shadows moderately thickened and fused. Lung fields generally clear. No thyroid or thymus shadows visualized. Heart and vessels normal. Conclusions negative.

Urinalysis May 13th: Cloudy, acid. Sugar negative, albumin negative, microscopic negative.

Blood May 13th: Total W. B. C. 8,900; hemoglobin 70%; R. B. C. 3,600,000. Culture nose negative K. L. Throat negative K. L. Right ear, gram positive staphylococcus. Blood typing III.

Temperature ranged from 100.8 R to 103.8 R. No chills.

May 15th, 1930: Pale pink macular eruption over lower abdomen and left leg. Eye grounds normal. Entire spine rigid. Incontinent.

X-ray of right mastoid shows destruction of trabeculae and general clouding; left mastoid negative.

Head held to right. Pupils normal in size and normal in reaction to light. Brudzinski positive. Babinski negative. Oppenheimer negative. Chaddock negative. K. K. normal.

Left ear negative. Throat and nose negative. Drum membrane right is flat; serosanguineous discharge. Smear shows no capsule organism.

Culture from ear, staphylococcus albus. Interne unable to obtain blood for culture. Immediate exenteration of mastoid urged. Temperature 101 R. to 104.8 R.

Diagnosis: (1) otitis media, right; mastoiditis right; (2) meningitis, cerebellar involvement right (?).

5:00 p. m. Spinal puncture and cisterna magna puncture failed. Operation not permitted on this date.

May 16, 1930: Left pupil larger than right. Left ear negative. Incision in right drum membrane adequate. Scant discharge right. Operation urged. Transfusion suggested. Temperature 100.8 to 103.8 R. Lumbar puncture (bloody).

Schilling count: Myelocytes 2 per cent, stabs 17 per cent, segments 14 per cent, lymphocytes 57 per cent, monocytes 10 per cent.

3:00 p. m. Unsuccessful attempt to give blood transfusion.

Operation: Local anesthetic. Simple right mastoid exenteration with exposure of dura of posterior fossa (normal). Necrosis and serosanguineous fluid in mastoid. Pale granulations here and there. Culture from mastoid, gram positive staphylococcus.

Just prior to operation there was horizontal nystagmus to the right when looking to the right (first degree); but only when patient was turned to the right. This nystagmus was accompanied by a tendency to conjugate deviation to the left. Immediately after the operation there was no longer any nystagmus when patient was turned to the right. Forty-five minutes later the child turned the head from side to side and

when the head was toward the right there again appeared a horizontal nystagmus (large, slow excursions). The tendency to conjugate deviation was more marked after the operation than before.

5:45 p. m. Patient flat on back with face turned upward toward head of bed. Horizontal nystagmus to the right.

May 17th, 1930. Nystagmus entirely absent even when patient is turned quickly to right. Pupils equal and react normally to light. Suggestion of a bilateral Kernig. Brudzinski positive with right leg. Less neck rigidity. Patient is pale, alert. Does not cry out as much. Temperature 103.2 R.

2:00 a. m. 102.8 R.; 12:00 m., 104.6 R. Nourishment well taken. Pulse of good quality. Transfusion deferred as the consensus of opinion was that its value was very uncertain and the past difficulties in finding a vein made those in attendance fearful of the shock; the same applied to spinal puncture.

Convergent strabismus.

May 18th, 1930. Patient was much brighter, neck rigidity less marked. No nystagmus, K. K. equal. No Kernig. Heart and lungs negative.

Later, general convulsions.

Has less pain apparently. Pulse good quality. First mastoid dressing. Wound appears to be in good condition. Takes nourishment well.

May 19th, 1:30 a. m. Convulsions, general. Patient somewhat cyanotic. Twitchings of face. No spontaneous nystagmus. Conjugate deviation of eyes to the right after convulsions.

Later: Brudzinski positive right. Less neck rigidity. Pupils normal in size and react to light. Patient follows normally. No nystagmus upon turning patient to right.

P. M.: Brudzinski positive with right leg. No nystagmus when turned to right. No facial twitching. Wound dressed. External canal dry.

May 20th, 1930. The left ear remains negative in appearance. Throat negative. Less neck rigidity. No nystagmus upon turning the patient in bed. Temperature 102.6 R. to 103.6 R. Restless and moaning. Fontanelle, anterior tense.

May 21st, 1930. Wound looking well. No nystagmus upon turning in bed. Chest clear. Temperature 101.8 R. to 102. Eyes fix upon an object, stares two minutes, then eyes slowly cross.

Pupils well dilated, media clear. Discs normal (color and outline). Vessels not enlarged, not tortuous. No hemorrhage nor exudate. Summary, normal fundi.

5:30 p. m. Rotary nystagmus to right and some nystagmus with quick component to left. Patient somnolent. Eyes roll about.

7:30 p. m. Coughing a little, breath sounds in upper lobes are harsh and there is a definite impairment of resonance in the left apex, anteriorly and posteriorly. No definite râles. Brudzinski and Kernig negative. Eyes are very unsteady and conjugate movements are poorly carried out.

8:20 p. m. Patient looks worse. Pays no attention to her surroundings. Anterior fontanelle pulsates and is soft. Pupils dilated. There is a horizontal nystagmus to right and left. Lungs P. N. everywhere resonant, B. S. vesicular. No râles. Heart sounds clear. No murmurs. Abdomen: no masses or tenderness. Neck slightly more rigid than on 18th.

No Kernig. No Babinski or Brudzinski. Impression is that of a process in the cerebellum, most probably an abscess. Eyes roll to right, then cross.

May 22nd, 1930. Facial paresis on right, convergent strabismus. Conjugate deviation to right, horizontal nystagmus to left, almost no neck rigidity. Brudzinski right and left, slightly positive. There is constant movement of left arm and left leg. The whole picture is of an extension of involvement to the right cerebral area. Left drum membrane normal.

9:00 a. m. Patient in extremis. Automatic movements of a stereotyped nature of left arm and left leg. Marked conjugate deviation with fixation of eyes to right. Fundi rather red but discs cannot be satisfactorily seen. Paresis of right side of body (face, arm, leg). Absent abdominals on the right.

Neurologist's diagnosis: Brain abscess left cerebral cortex, probably with an associated meningitis.

Nystagmus to the left. When the automatic movements of the left hand and leg take place the eyes tend to remain to the left. This conjugate deviation to the left is not as frequent as that to right.

Marked conjugate deviation of eyes with fixation to right. Fundi red, discs slightly blurred. There is paresis of right side of body (face, arm, leg). Absent abdominals on right, present left. Babinski right. Probably brain abscess, left cortex, with associated meningitis. Lumbar puncture: cell count 7,800 polymorphonuclears, no intracellular organisms. Cisterna puncture, cloudy fluid. Culture, spinal fluid, no growth.

Operation (by Dr. R. Klemme). $\frac{1}{2}\%$ novocain. Incision over left motor cortex. Opening made with perforator and burr. Needle inserted in all directions in the hope of striking an abscess. None was found. Right mastoid wound reopened and the cerebellum explored; struck a dilated cisterna lateralis; no evidence of an abscess.

Following return from operating room, patient had a series of convulsions; the twitching being present more in the right arm and leg than on the left. Right side of face twitching almost continuously. Eyes rotating continuously. At 2 p. m. the twitching stopped and the nurse reported movements of the right arm. Pulse strong, 108. Color of patient very good. Shortly after this, patient became somnolent, color poor, pulse very rapid. Temperature 104, pulse 180 (with stethoscope), respiration 48. Left arm and leg moving.

May 23rd, 1930. Patient very pale; lies quietly on back. No movement except of respiration and left foot slightly. The eyes are in conjugate deviation to right most of time. Occasionally they move slowly to left as far as midline, then back to right and not together. No definite neck rigidity. Patient is moribund. Right side of face cold and pale. Unable to swallow.

Marked spasticity of all extremities. Anterior fontanelle still soft.

May 24th, 1930. Eyes divergent and fixed; left eye up and to left, right eye up and to right. Pulse almost imperceptible. Entire body seems rigid.

One hundred fifty c.c. saline subcutaneously. Karo and orange juice into stomach, 10 p. m. Some improvement after forcing fluids throughout forenoon but at 2:30 pulse became very weak and patient had a series of convulsions, the twitching being almost entirely on the left side but the

right side showed some movement. Eyes remain open. Periods of apnea from 3:30 on.

May 25th, 1930. Caffeine and O₂—CO₂ mixture administered. Both pupils dilated, more right. Dilatation of nostrils with inspiration. Constant expiratory grunt. Eyes open constantly; right pupil dilated more than left. Vomiting. Temperature 107.2 R. Respiration shallow.

2:03 p. m. Patient died.

Pathologist's report on bone from mastoid, right: Bone necrotic. Some polymorphonuclear leucocytes and eosinophiles are seen within the increased connective tissue.

Autopsy: Diffuse leptomeningitis. Purulent exudate over entire cortex and extending over base and stem. Ventriculitis (right, lateral). Pus in right labyrinth. No brain abscess either of cerebrum or cerebellum (Dr. Gray).

SUMMARY.

The patient was well until May 11, 1930. At this time she became feverish and vomited. On May 12, 1930, the family physician found an otitis and the patient drowsy. A myringotomy was performed on the right by him. On May 13, 1930, upon entering the hospital the patient was irritable, had neck rigidity and a temperature of 102 R. There were shrieking cries when awake and asleep. Undoubtedly there already existed a suppurative meningitis. It is greatly to be regretted that the caloric test was not made. The only possible reason we can, in retrospect give, is that inasmuch as the extension intracranially already existed, the caloric test no longer seemed important. The Schilling count of 17 per cent stabs with only 14 per cent segments (total W. B. C. 8900, 8500) showed a severe infection with little resistance. The conjugate deviation of eyes to right after convulsion on May 19th spoke for a cerebral cortex involvement on the right, just as the conjugate deviation of the eyes to the left previously spoke for a left cortical involvement.

The constant movement of left arm and leg on May 22nd spoke for an irritation of right cortex. The paresis of the right side of the body the neurologist interpreted as a sign of possible abscess of the left cerebral cortex, probably with associated meningitis.

The dilated right cisterna lateralis ruled out a cerebellar abscess on that side.

OBSERVATIONS ON MICROSCOPIC SECTIONS—WOLFF.

The microscopic sections were prepared in the routine manner. They were sectioned at twenty micra and stained in hematoxylin and eosin. The left ear was cut in vertical plane and the right in horizontal plane. It will be recalled that the left ear, which shows the greater degree of labyrinthitis, was the unoperated side.

Left Temporal Bone.—The dura over the mastoid portion of the left temporal bone is highly edematous. Numerous small thrombi of the mural type appear both in small veins and arteries of the dura. In the mastoid bone proper a cartilaginous area still persists inferiorly. Surprisingly long trabeculae of bone jut into the antral cavity. The borders of the trabeculae are somewhat ragged and irregular. Water marking is present. Where the bone is ragged the alignment of osteoblasts is lost but no typical osteoclasts can be seen.

The lumen of the antrum appears free from pus when examined with the unaided eye. When seen under the microscope, however, a slight amount of pus may be seen in a few of the para-antral cells. Section 260 shows numerous coccus bacteria in clumps. The submucosa in the posterior part of the antrum is not at all thickened. The epithelium is normal. The blood vessels in the submucosa are tremendously engorged, and diapedesis has occurred in numerous areas. Polymorphonuclear leucocytes, where seen in the submucosa, are in definite alignment and appear to be confined within tiny vessels. As one approaches the middle ear cavity proper, the pus in the para-antral cells becomes more abundant, the submucosa is infiltrated and the epithelium is lacking. Hemorrhages are more frequent. In some areas (section 490) the submucosa is eroded to the basement membrane. In others there are areas of granulation tissue. As may be observed in Fig. 4, Sec. 580, the facial nerve is lacking in the facial canal. This disappearance of the nerve exists between sections 390 and 680. Section 470 shows an aperture in the wall of the facial canal, but there is no evidence of infection entering from the middle ear cavity. The venous plexus surrounding the facial nerve is filled with pus. There is, however, no evidence of a pathologic degen-

eration of the nerve fibers where they reappear. The only explanation of the condition that can be offered is the following: The facial nerve had become edematous with the progressive course of the meningitis and a concomitant softening of the fibers of the nerve occurred. When the bone was removed at autopsy probably the nerve was not completely severed at either end of its course. A violent pull in the effort to remove the petrous part of the temporal bone from its firm position could easily have pulled apart at its weakened point a nerve already softened from edema. The present state may therefore be considered as an artefact.

The secondary tympanic membrane shows congested vessels, especially on the middle ear side. Infiltration has occurred both on the middle ear and cochlear side. A plug of granulation tissue is present in the niche of the round window.

The eustachian tube is filled with pus. A heavy mononuclear infiltration is present in the submucosa, and this tissue appears cystic. Polypoid formation is present and the ciliated epithelium is markedly sloughed off. It appears more abnormal toward the pharyngeal end than toward the middle ear orifice.

The most striking feature in the pathologic condition of this ear is the great abundance of pus in the labyrinth as compared with the amount in the middle ear. In certain areas the pus is confined in the endolymphatic system. See Fig. 4, Sec. 580. The maculae sacculi et utriculi are both badly eroded. Fig. 5 left, Sec. 580 detail, shows the condition in the region of the ampullae of the horizontal and superior canals. These two canals, even in their most distal curvatures, are densely filled with pus, particularly in the endolymphatic spaces.

The vessels in the perilymphatic tissues are greatly congested. In the vestibule pus appears in the cisterna. At the level of the oval window the pus is much more abundant within the vestibule than on the middle ear side where an attempt at healing has occurred. (Fig. 4, Sec. 580; Fig. 6, Sec. 580; Fig. 7, Sec. 610.)

The organ of Corti is completely degenerated in all turns. Masses of pus are seen in all scalae. Reissner's membrane has been destroyed in many places. Abundance of pus is seen in the perineural and perivascular sheaths of the modiolus. The internal auditory meatus is also filled with purulent substance.

RIGHT TEMPORAL BONE NO. II.

The right temporal bone is exceptionally interesting because of the fact that here is a case upon which an operation has been performed.

An interesting point of contrast is seen in the mastoid region of the two ears. The well pneumatized condition seen on the left in Fig. 4, Sec. 580, as well as sections 90-460 (not illustrated) indicate that this infant had the well pneumatized type of antrum. Of course much marrow still persists in the apical and para-antral cells, as is to be expected in an infant of this age; but cavitation of the mesenchyme is quite complete. The only evidence of its previous existence is a somewhat thicker submucosa than is commonly found in normal older specimens. Fig. 16 right, Sec. 420, taken from the right side, shows a plug of mesenchymatous looking tissue forming a web across the cells in this mastoid antrum. It is difficult to prove that this is not true mesenchyme, but it is the opinion of the authors that this is young scar tissue. The foundation for this opinion is based upon the following observation: The very definite border line of the mucosa of the para-antral cells extends right through this tissue. If the tissue filling the space were homogeneous and in complete continuity with the web in the center it might well be a mesenchymal remnant. Since it is not continuous, it must be cicatricial tissue. The same is true of that in the oval and round windows in this case. The continuity of the tissue as seen in one section alone cannot be considered as proof of the presence of mesenchymal tissue. Serial sections must be studied. If in every one of the series the webs are always homogeneous with the submucosa, the tissue is certainly mesenchymal.

The dura over the right mastoid has been stripped off at autopsy. In the mastoid bone proper, as on the left, cartilage may be seen. There is evidence of more active bone growth on this side. No long trabeculae of bone may be seen here. They probably have been shattered at operation. Those present are even more ragged and water marked than on the opposite side. Many of the air spaces are filled with young scar tissue and granulation tissue as noted above. The submucosa is infiltrated with both

mononuclears and polymorphonuclears. In places the mucosa and submucosa are torn off down to a layer of rather dense connective tissue. This may be due to the mechanical disturbance at operation. Masses of pus are seen in the para-antral cells. Numerous large eccentric mononuclears are present. In the middle ear proper the condition appears somewhat more chronic. The niche of the oval window is well filled with scar tissue. Unfortunately, this ear was badly shattered at autopsy. The footplate of the stapes has been forced into the vestibule and the contour of the oval window destroyed. (Fig. 8 right, Sec. 400.)

The eustachian tube (see Figs. 14 and 15) contains much pus on this side also, but the ciliated epithelium, except for certain patchy areas, is in much better condition than that of the left. Polypoid formations are present.

The labyrinth on this side is not nearly so heavily laden with pus as on the opposite side. The same type of huge eccentric monocytes found in the middle ear are also present in abundance in the inner ear. Here they appear in tufts on the membranes in fungus-like growths. They occur principally within the endolymphatic system in the vestibule, but in the cochlea they are in the scala vestibuli and tympani rather than in the scala media. Although a certain amount of pus probably entered the labyrinth through the shattering of the promontory at autopsy, it is evident that there was labyrinthine involvement before death. The component cells of the organ of Corti are indistinguishable, save perhaps the pillar cells. The organ is, however, in better condition than that of the left. (Compare Figs. 9 and 10 with Figs. 2 and 3.) The tectorium is lacking throughout, although vague remnants are in the apical turn. The endolymphatic duct does not appear to have been an avenue of infection either to or from the inner side, as its lumen appears clear throughout. The internal auditory meatus as on the left is filled with pus (Figs. 11 and 12).

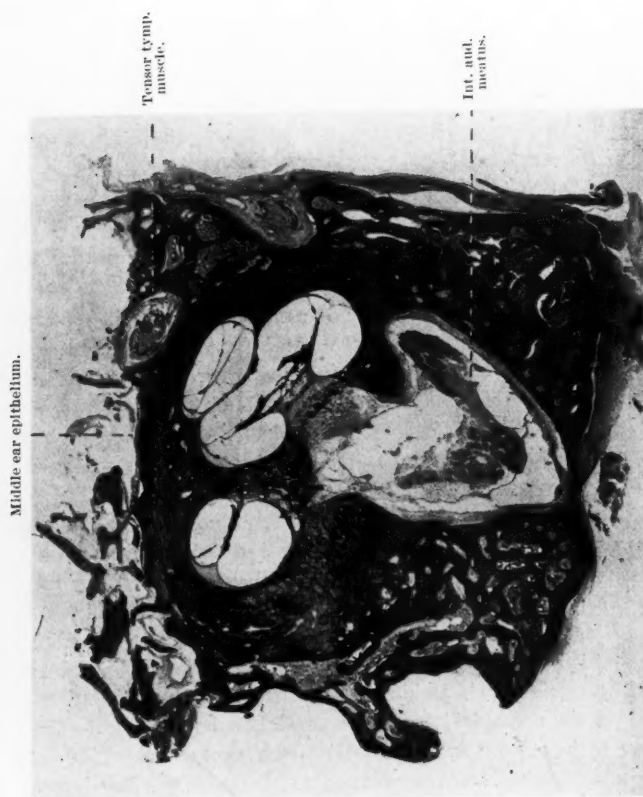


Fig. 1. No. II, Left, Sec. 830. Vertical cut showing destruction of organ of Corti in all turns, internal auditory meatus containing pus, middle ear epithellum sloughed off.

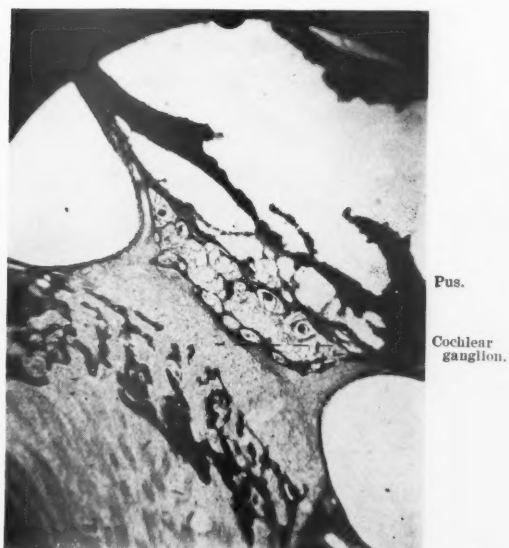


Fig. 2. No. II, Left, Sec. 830. Heavy accumulation of pus in cochlea (Detail of Fig. 1).

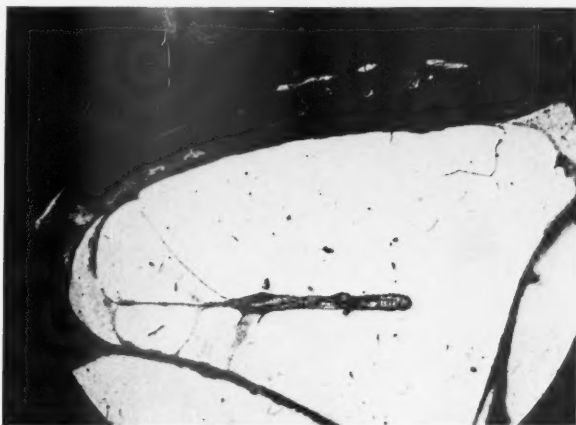


Fig. 3. No. II, Left, Sec. 580. Apical turn. Organ of Corti entirely obliterated. Compare with Fig. 10, Right, Sec. 220.



Fig. 4. No. II, Left, Sec. 580. Showing pus primarily in the endolymphatic system, although it is also in the cisterna. This section also shows the absence of facial nerve.



Fig. 5. No. II, Left, Sec. 580. Detail showing a cut completely across the crista of the ampulla in the posterior canal. It is surrounded by pus.

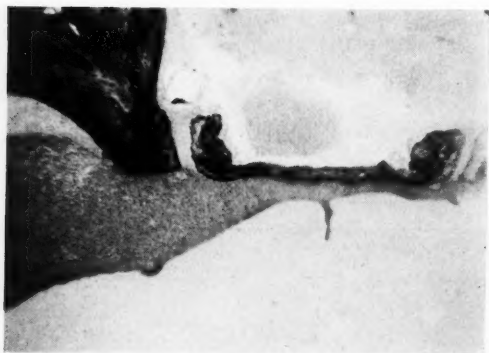


Fig. 6. No. II, Left, Sec. 580. Pus in cisterna. Stapedial ligament still intact.

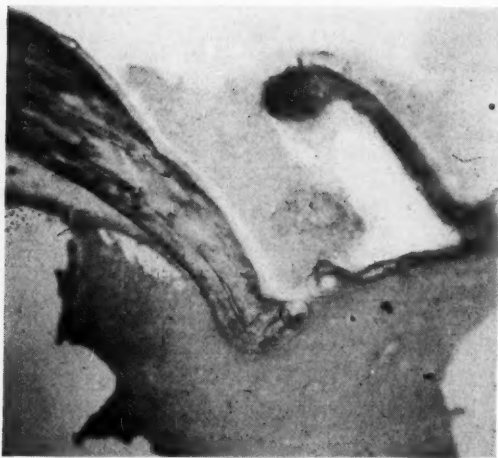


Fig. 7. No. II, Left, Sec. 610. Showing relatively heavier grade of pus in inner ear than in middle ear.



Fig. 8. No. II, Right, Sec. 400. Horizontal section showing basal turn where it enters the vestibule. The specimen was somewhat crushed at autopsy.

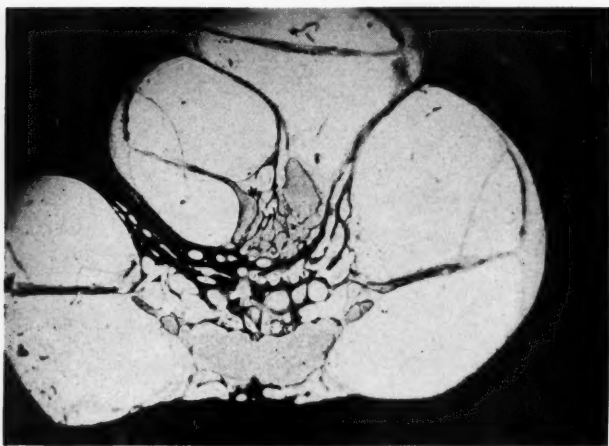


Fig. 9. No. II, Right, Sec. 220. Contrast the condition of this right cochlea (operated side) with the left (Fig. 1).

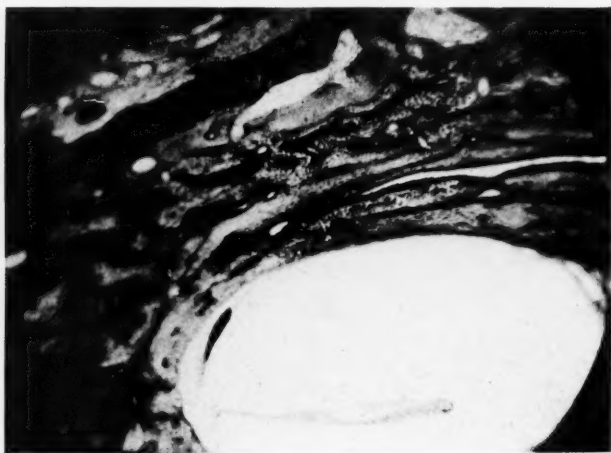


Fig. 10. No. II, Right, Sec. 220. Apical turn. Organ of Corti in fair condition. Calcium deposit along stria vascularis.



Fig. 11. No. II, Right, Sec. 400. Heavy infiltration of pus along the vestibular nerve but the macule of the sacculus is still in good condition.



Fig. 12. No. II, Right, Sec. 400. Pus in internal auditory meatus near the vestibular ganglion cells.



Fig. 13. No. II, Right, Sec. 420. The anterior part of the annular ligament has been torn at autopsy. Pus and cicatricial tissue may be seen.



Fig. 14. No. II, Right, Sec. 270. Eustachian tube. Cystic submucosa, polyp formations, fairly normal columnar ciliated epithelium.



Fig. 15. No. II, Right, Sec. 270. Detail of mucous membrane of E. T. Note cystic submucosa.

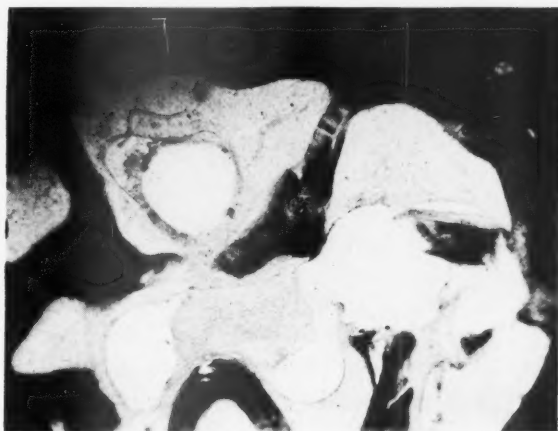


Fig. 16. No. II, Right, Sec. 420. Showing web of young scar tissue in para-antral cell of operated side.

XIV.

CAVERNOUS SINUS THROMBOPHLEBITIS FOLLOWING TONSILLECTOMY.—REPORT OF A CASE.*

EDWARD H. CAMPBELL, M. D.

PHILADELPHIA.

Septic thrombophlebitis of the cavernous sinus following tonsillectomy is an extremely rare complication of this operation as is evidenced by the very few cases reported in the literature. Cases of infection of this blood sinus from a primary focus in the face, nose and accessory nasal sinuses have been rather commonly observed and recorded but practically no mention has been made of cases following operations upon the throat.

Mainzer¹ has collected a few statistics on its occurrence in general. In a review of the literature in 1930, he found approximately 350 cases recorded. He reports Chisholm and Watkins as having found 8 cases in 50,000 admissions on the surgical service at Johns Hopkins Hospital. Also that the New York hospitals reported 21 cases in 80,000 admissions and the Clearfield Hospital 2 cases in 15,000 admissions. Mainzer further states that the foreign literature reported 19 cases and this country 21 cases since 1917. He cited Dorland Smith's review of 140 cases, 40 per cent of which were secondary to ear infections; 13 per cent to mouth and throat infections; and 9 per cent to nasal infections.

The great majority of those cases following mouth and throat infections have been secondary to abscesses in the peritonsillar or retropharyngeal regions, and the cases following tonsillectomy will be found to have first developed infection at the site of the operation.

REPORT OF A CASE.

The following is an example of the serious complication that may follow the removal of tonsils:

*Read before the Section on Otolaryngology of the College of Physicians of Philadelphia, October 21, 1931.

From the Department of Otolaryngology of the University of Pennsylvania.

Mrs. S., age 35, was admitted to the Lankenau Hospital Nose and Throat Dispensary for tonsillectomy on October 13, 1930. Under local anesthesia by infiltration around the tonsils with one percent procain solution containing a few drops of adrenalin, her tonsils were removed by the dissection and snare method. There was some bleeding immediately following the operation but this did not require ligation or suturing for its control. She was not discharged from the hospital on the following day, as is usual with these patients, because of a temperature of 100° F. No other unusual symptoms were present at this time. On the next day, however, she developed a severe chill followed by a rise of temperature and a swelling in the right peritonsillar region suggesting an early abscess. This abscess developed rapidly in the next few hours and on the following day was incised with the evacuation of thick greenish colored pus. Examination of this pus revealed the presence of gram positive staphylococci and gram positive diplococci. Her temperature that day varied from 99.4° F. to 100.3° F. Her leucocyte count was 17700 with 82 per cent neutrophiles.

Following the incision of the peritonsillar abscess the throat symptoms improved but the next day she developed pain and tenderness in the left temporal and parotid region. Within the next two days there developed an edema of the eyelids, first of the right eye and shortly after the left. In addition, the tenderness and swelling of the left side of the face increased and extended downward over the parotid region but no fluctuation developed. There was also some swelling in the left peritonsillar region but not sufficient to indicate an abscess in that region. She had a daily chill with rise of temperature following it but only to a moderate degree. Blood culture taken at this time showed the presence of nonhemolytic streptococci. During the next two days the swelling of the eyelids rapidly increased and a distinct proptosis was evident. Eye examination showed paralysis of the right external rectus with other ocular movements faulty but no swelling of the optic discs nor hemorrhages. On October 23, ten days after operation, the swelling of the left side of the face had developed to an extent where it was thought wise to incise, although no distinct fluctuation was evident. Deep incision in the pre-

auricular area released a small amount of pus and very dark colored blood. On account of the small amount of pus obtained this incision was enlarged and deepened on the following day but no abscess pocket was found. Culture of this pus revealed gram positive streptococci.

During this time the ocular signs of cavernous sinus thrombosis had steadily progressed. Both eyelids became markedly edematous, the exophthalmos steadily increased, considerable ecchymosis developed and the eyeball itself became immovable.

From the onset of her illness the patient had been treated by general stimulation, frequent infusions of glucose and saline and by blood transfusions but she grew steadily worse and on October 26th died, with a temperature of 107° F., on the thirteenth day following the operation.

Only a partial autopsy could be obtained. The incision on the left side of the face was explored and a considerable amount of seropus was expressed from the woody tissues but no localized pocket could be found. Sections of the lungs showed multiple hematogenous abscesses, the heart and liver showed cloudy swelling and the kidneys gave evidence of acute tubular necrosis. Unfortunately the brain could not be obtained but the typical signs and symptoms of cavernous sinus thrombophlebitis indicated plainly the diagnosis.

COMMENT.

This patient represents an unfortunate sequence of events that may perhaps be traced to the injections of the local anesthetic preparatory to the tonsillectomy. Her tonsils were of the badly infected type with open crypts containing considerable cheesy exudate and probably pus. It is easy to imagine the needle penetrating this infected material and carrying organisms into the peritonsillar space where the abscess subsequently developed. It is quite possible that any peritonsillar abscess, originating spontaneously, could give a similar result but in this operated case there is the influence of not only the needle point, which may have injured a venous wall, but also the traumatizing effect of the tonsillectomy and the efforts thereafter to control the bleeding. We can assume that there developed here a septic thrombophle-

bitis of the veins of the pterygoid plexus adjacent to the peritonsillar abscess coinciding with the chill on the second day following operation.

The process of extension of the infection from here to the cavernous sinus may be in three different ways. It may extend by a retrograde thrombosis up through the small emissary vein communicating directly with the cavernous sinus or through the branch from the inferior ophthalmic vein, which in turn runs into the cavernous sinus. Secondly, there may be extension of the septic thrombosis directly into the jugular vein and from here by a retrograde thrombosis up through the jugular bulb and inferior petrosal sinus to the cavernous sinus. This is the most commonly accepted method of the formation of a cavernous sinus thrombosis from the throat area. The third process of extension is by the dislodging of a septic embolus from the pterygoid plexus which could take the route through the deep facial branch to the facial vein. From here it is necessary to assume a reversed current of blood up the valveless facial vein, angular and ophthalmic veins into the cavernous sinus. Here the septic embolus could be caught in the fibrous network interlacing this sinus and go on to the development of a thrombophlebitis.

It would seem that this latter route is the one that is likely to have been followed in this case as the rapidity with which eye symptoms developed following the initial chill would preclude the transmission of the infection by the slower method of retrograde thrombophlebitis.

The infection on the left side of the face can be accounted for by the development of a left peritonsillar abscess situated deep and penetrating the superior constrictor muscle to involve the parotid gland. The multiple abscesses found in the lungs are what would have been expected from the septic emboli in the general circulation.

Such a case as this should emphasize the care with which a local tonsillectomy should be performed. Thrombosis of the peritonsillar veins following this operation is the usual process, as was proven by Fetterolf and Fox², who examined the peritonsillar regions of dogs on whom they had performed tonsillectomy. They

found numerous thrombosed vessels not only on the surface of the operated area, but also deep in the peritonsillar tissues. If infective material is carried into this thrombotic area with the anesthetizing solution, a thrombophlebitis with its serious sequelae is very likely to occur.

2117 CHESTNUT STREET.

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XV.

IMPORTANT POINTS IN FOUR THEORIES ON
OTOSCLEROSIS.*

EDMUND PRINCE FOWLER, JR., M. D.,
NEW YORK.

There are so many misunderstandings about what various leading authorities believe in regard to the histopathology and etiology of otosclerosis that a resumé of some of the more important points would seem to be of value. The ideas of Brühl, Mayer, Weber and Wittmaack are perhaps the most outstanding and certainly they seem to be at greatest variance with each other. Furthermore, through letters from Dr. Arthur B. Duel and my father, I have had the rare privilege of meeting them and discussing the subject with them personally. I should like to take this opportunity to thank them publicly for their cordiality and hospitality and also their generosity in providing me with the material for this article and the exhibit for the Research Council of the American Otological Society. Actual specimens and lantern slides provided by these men and by Prof. Von Eicken of Berlin, Prof. Ruttin and Dr. Brunner of Vienna are on exhibition in the rooms of the Central Bureau of Research of the Academy of Medicine, New York City.

In presenting the ideas of these men I should like it distinctly understood that I may be misinterpreting them. I shall try to present the work of each as he individually would like to have it presented, but for a full and accurate account their original articles are bound to be more reliable.

GUSTAV BRÜHL.

Professor Gustav Brühl¹ of Berlin summarizes his beliefs on the subject of otosclerosis with five points:

*Presented before the annual meeting of the American Otological Society, at Briarcliff Lodge, June 18-20, 1931.

From the Department of Pathology, College of Physicians and Surgeons, Columbia University, New York City.

1. Clinical otosclerosis is identical with osseous ankylosis of the stapes.
2. For proper investigation of otosclerosis more cases of ankylosis of the stapes diagnosed *in vivo* should be studied before many conclusions are drawn on the subject.
3. Otosclerosis is a constitutionally conditioned, hereditary affection, a kind of degeneration or atavism, but not a disease. Therefore, it is idle to look for its cause as for the cause of a disease.
4. Not all departure from the normal labyrinthine bone structure should be classed as otosclerosis or incipient otosclerosis.
5. The true pathologic bony form leading to ankylosis of the stapes consists of a spongelike, new formed bone which starts from the spaces surrounding the periosteal blood vessels in front of the vestibular window. By mechanical (or chemical) irritation there is a disturbance of the old bone by resorption and later a replacement by new bone. The new bone is formed in excess and

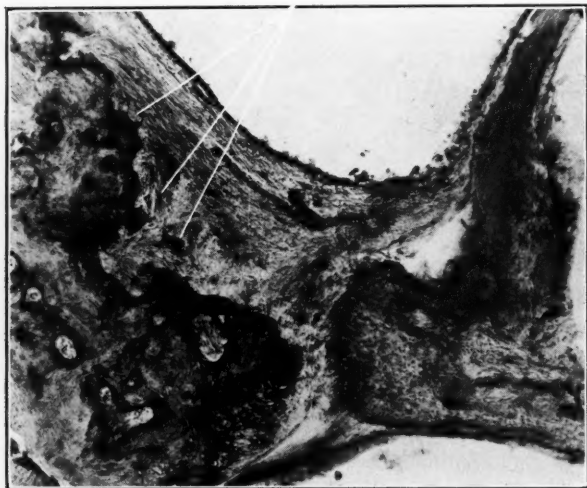


Fig. 1. Hyperostoses in a case of otosclerosis. (Specimen prepared by the author in the Department of Pathology, Columbia University.) Magnified $\times 100$.

is therefore to be recognized as tumorlike new formed bone. Between the most anterior portion of the tensor tympani tendon and the anterior border of the annular ligament lies a portion of the cochlear wall, dubbed by Brühl the "otosclerotic corner." In the adult, in contrast to the embryo, the anterior portion of the annular ligament is broader than the posterior portion.

The irritation of the movement at this point, where the foot-plate moves farthest and the pull of the tensor tympani in the opposite direction, Brühl² believes to be factors in the production of otosclerosis. This is especially the case in individuals hereditarily predisposed to the hyperostoses which often form in this region. The stress and strain of the pulling and the irritation of the hyperostoses together are, in his opinion, most important in the causation of otosclerosis. (Fig. 1.)

KARL WITTMACK.

The ideas of Professor Wittmaack of Hamburg,^{3 4 5} are at considerable variance with those of Professor Brühl. He believes that venous stasis causes otosclerosis. He bases his theories on the growth of the process on the various phenomena observed in a similar disease, which he has been able to produce in the labyrinthine capsule of chickens by venous stasis. He thromboses the sinus which drains the capsule of the hen with concentrated iron chloride. The result is a disturbance in the bone which Wittmaack believes to be identical with that found in human otosclerosis.

He divides the process into several stages. In the first few days after the operation there is simple halisteresis (removal of calcium from the regions surrounding the blood vessels). Next there is resorption or widening of the blood vessel channels (Fig. 2), produced by pressure of the vessel itself or by osteoclasts. A reaction now sets in and there is rebuilding of two kinds: (1) further growth and laying down of fibers of the cells of the old bone between the resorption spaces, and (2) filling of the resorption spaces with lamellar bone. Even in the hen the process is not always as simple as in the case just described, for, as a rule, all of the processes go on at the same time, and when seemingly complete may start all over again with halisteresis, resorption



Fig. 2. Experimental chicken otosclerosis, resorption stage. (Specimen prepared by Karl Wittmaack, Hamburg. Photo by author.) Magnified $\times 150$.

Note: This photograph does not do justice to the similarity between the lesion shown and human otosclerosis. It does, however, show the large marrow spaces and engorged vessels.

and further back building. This last jumbled picture is what is ordinarily seen in human otosclerosis. Only in worn out processes do we have one stage, the completed rebuilding, the so-called healed stage, consisting of a compact many celled bone often with well marked lamellar systems.

Wittmaack believes the otosclerotic bone to be continuous with the surrounding bone. It has areas which have been resorbed and in the late stages a few areas which may have been replaced by lamellar bone, but primarily the foci consist of altered old bone and not of new bone at all. He believes that a somewhat warped framework always remains and that there is no cement line about a focus of otosclerosis, such as is always found around new formed bone, except at the rare points where lamellar bone happens to have been formed along the edges.

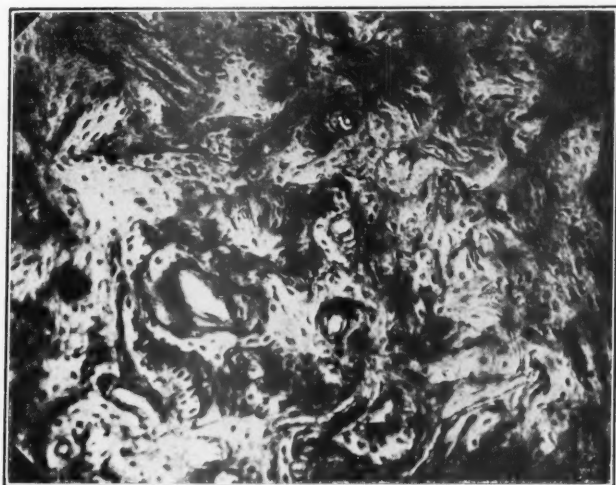


Fig. 3. Normal labyrinthine capsule of the chicken. (Specimen prepared by Karl Wittmaack, Hamburg. Photo by author.) Magnified $\times 150$.

Wittmaack has a very strong position, in that his beliefs are based on controlled phenomena (Fig. 3) in his operated hens. In one day he produces his "halisteresis," in four or five days he can demonstrate his resorption, in two or three weeks after the operation he can begin to demonstrate the beginning of "back-building." To further confirm his theory that venous stasis is the etiological factor of greatest importance in otosclerosis he has demonstrated in a few cases a greatly engorged blood vessel leaving the otosclerotic focus in humans. Wittmaack¹⁶ has now published a demonstration of the exact point at which the stasis takes place.

The big question is whether what Wittmaack produces in the hen is really otosclerosis or not? A set of colored photographs, which he very kindly sent me, illustrate how very closely the experimentally produced phenomena in the hen simulate those found in human otosclerosis. (Fig. 4.) These were demonstrated at the Collegium and were on exhibition at the Academy of Medicine, New York City.

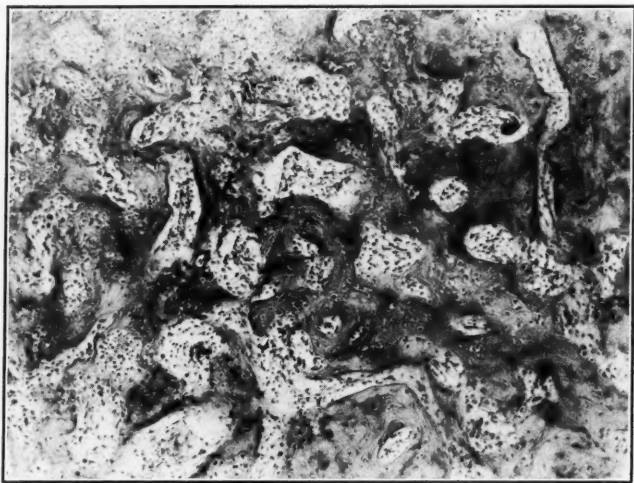


Fig. 4. Human otosclerosis in the resorption stage. Compare Fig. 2. (Specimen prepared by Karl Wittmaack, Hamburg. Photo by author.)

MORITZ WEBER, JR.

Dr. Moritz Weber, Jr.,⁶ read a paper before this Society last year, in which he expressed the opinion that otosclerosis may be regarded as a localized osteitis, fibrosa, or, as he preferred to call it, a localized "osteodystrophia fibrosa." (Fig. 5.) He shows how very similar are the lesions in this disease with the bone processes in otosclerosis. Further, he shows what he considers to be identical lesions in the labyrinthine capsule of dogs⁷ which have been fed on a low calcium diet with no vitamin D (an osteoporotic rather than a rachitic diet). Weber has been working in Wittmaack's laboratory and is more or less in accord with the theory of the Professor. He points out that osteitis fibrosa is also produced by a venous stasis.

At the time that I was in Hamburg, Weber did not, however, believe that typical foci of otosclerosis are an altered old bone. He projected the idea of studying the fibers at the edge of otosclerotic foci with polarized light so as to make certain that they did or did

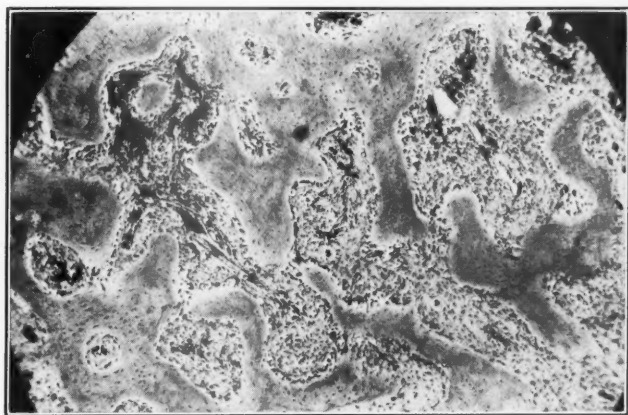


Fig. 5. Osteodystrophia fibrosa (osteitis fibrosa) in the dog. Specimen prepared by Moritz Weber Hospital Foundation. Photo by author.

not pass through the border of the focus. This he has now done, and the article is to appear shortly.⁸ He demonstrates a sharp demarcation between the fibers of the focus and the fibers of the normal capsule and furthermore demonstrates a cement line all the way around the focus. These two facts strongly indicate that the otosclerotic process is all new bone, a point which is most important and always causes a great deal of discussion whenever the histology of otosclerosis is being considered.

OTTO MAYER.

In order to follow the arguments used in the latest theory of otosclerosis, that of Professor Mayer of Vienna,^{9,10} one must first understand what he considers to be otosclerotic bone. For many years he has reiterated that otosclerotic foci are made up of short fibered weblike bone (geflechtartiger knochen),¹¹ which is always separated from the normal bone of the labyrinthine capsule by a cement line. It is quite difficult to be sure of this short fiber bone, and of the cement line in hematoxylin-eosin preparation, and much of the controversy over this point would be eliminated if differential stains were more universally used.

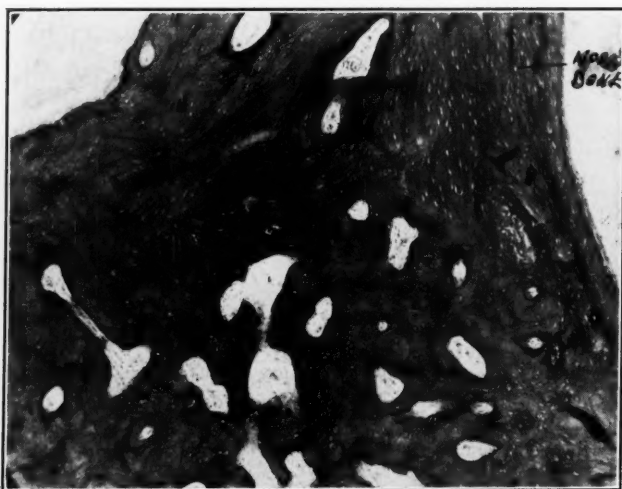


Fig. 6. Edge of otosclerotic area showing difficulty in seeing the border in a hematoxylin-eosin preparation. (Specimen prepared by Otto Mayer, Vienna. Photo by author.) Magnified $\times 50$.

Fig. 6 shows the border of a relatively old so-called "healed otosclerotic focus," much of the short fiber bone having been replaced. When lamellar bone is juxtaposed to lamellar bone, it is always difficult to be sure of the line of demarcation, but in this specimen it is even harder to be sure at the point where two different kinds of bone come together. (Wittmaack says of this very specimen that it is continuous; that the otosclerotic bone is not entirely rebuilt; that it is merely the old bone diseased.)

Fig. 7 is a section through the same temporal bone stained with silver and is approximately in the same region. Note that the line of demarcation between new lamellar bone of the focus and old lamellar bone is still difficult to see, but note now how sharp is the differentiation between some of the bone of the focus and the normal bone of the capsule.

This modification of the Bielchowsky-Marchi method shows what Mayer means by his "weblike" bone. There is some normality in the capsule, but Mayer believes it to be more abundant and



Fig. 7. Edge of otosclerotic area, modified Bielschowsky-Marchi method. (Prepared by Otto Mayer. Photo by author.) Magnified $\times 50$.

of a distinct type in otosclerosis predisposed individuals and points it out particularly in the region of the semicircular canals, between the vestibule and the first turn of the points of predilection for otosclerosis.

All this is an old story, but must be mentioned because it is precisely in these regions that Professor Mayer first found very striking, spontaneous and presumably nonsymptomatic fractures in the temporal bones of several cases of Paget's disease (osteitis deformans). It is well known that Paget's disease deforms the skull markedly and may even exert enough strain on the temporal bone to rotate it until the internal meatus opens upwards instead of posteriorly. Mayer finds his fractures very often in the promontory, near the anterior border of the oval window, and feels that from more careful study of the stresses we shall find this the weakest part of the bony capsule, and the part of the bone most subject to powerful mechanical influences. He does not say that otosclerosis is the callus of a healed fracture; rather he believes that a continuous strain on the primitive bone in heredi-



Fig. 8. Fracture through the promontory in a case of Paget's disease. Note connective tissue and regrowth of bone. (Preparation by Otto Mayer. Photo by author.) Magnified $\times 16$.

tarily predisposed individuals accounts for the growth of a more mature bone, otosclerotic bone. The fractures merely indicate the regions where stress and strain take place.

Fig. 8 is from a case of Paget's disease. Note the position and size of the fracture and the fact that it is filled with an organized connective tissue and even shows some regrowth of bone. It was the finding of fractures like this one which caused Mayer to search through his collection for similar evidences of mechanical influences on less obviously pathologic bones. (Fig. 9.) He found many, most not so large, to be sure, but they are none the less definite and all contain organized tissue. They are, therefore, not artefacts, as has been suggested.¹² He finds fractures mostly in old people or patients debilitated by disease, never in children.

Spontaneous fractures are not altogether a new discovery, for Nager, Manasseh, Brunner and others have described one between the vestibule or the niche of the oval window and the

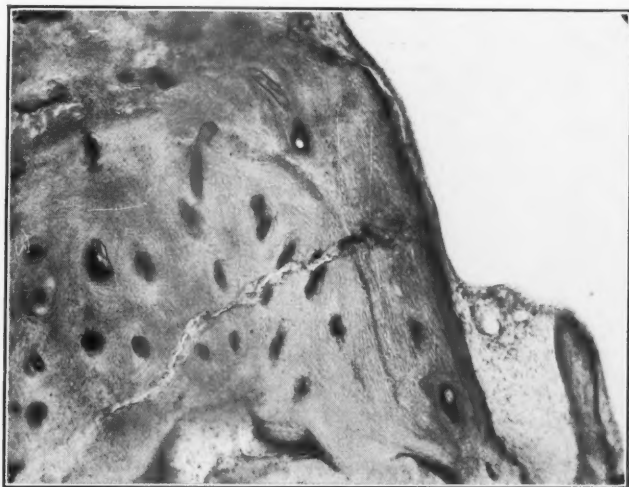


Fig. 9. Fracture through the promontory in a supposedly normal individual. (Specimen prepared by Otto Mayer, Vienna. Photo by author.)

posterior ampulla. Otosclerotic bone also occurs in this region, according to Mayer. Brunner¹³ has lately found many fractures similar to those found by Mayer. The findings of these fractures by no means solves the problem. Professor Mayer is now studying the framework of the bones of the skull, the brain, etc., in order to determine the mechanical influences responsible for them. As stated before, he does not believe that otosclerosis is a healing of actual fractures. He believes, to the contrary, that otosclerosis bone is like the bone found by Müller¹⁴ in the rarified regions of the points of greatest stress in cases of marked tibial bowing due to rickets. Such regions appear as fractures in the X-rays, but on section are found to be a loose short fibered weblike bone. They are very similar to callus and represent an attempt of immature bone to develop a bone capable of withstanding the demands made upon it. The most mature bone which can be formed is lamellar bone. This bone is sometimes formed in otosclerosis, but, as a rule, the bone is left in some intermediate

form between the very primitive bone of the normal capsule and a true lamellar bone.

CONCLUSION.

1. Brühl believes that otosclerosis is caused by the irritation of the several factors working at the "otosclerotic corner."
2. Wittmaack believes it to be caused by venous stasis.
3. Weber finds it identical with osteitis fibrosa and puts it on a local metabolic basis.
4. Mayer believes it to be a response to a profound strain, the nature of which he has not yet determined, but the presence of which he proves by the finding of fractures in the points of predilection for the disease.

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XVI.

ATROPHIC RHINITIS.*

JOHN A. PRATT, M. D.,†

MINNEAPOLIS.

In the latest ear, nose and throat book is stated that "Atrophic rhinitis is an atrophy of the nasal mucous membrane and the underlying soft tissues, eventually involving the turbinal bones. The etiology is unknown, as no pathologic condition or specific organism has ever been proven to be the causative factor." These same statements have been made in all papers and books written in the past to which I have had access.

Dr. Pollock, in his deductions from the paper of Dr. Vogel, and presented before the Middle Section of the American Laryngological, Rhinological and Otological Society, January 17, 1931, under the title of "A New Conception of the Etiology of Atrophic Rhinitis," states "The primary stage is a sinusitis (which exists in all atrophic cases), with subsequent infection and degeneration of the ganglion and thence leading to a similar degenerative change in its nerve fibers, which end in the nasal mucous membrane. As only a few atrophic cases result from nasal sinusitis, one must more or less assume the presence of predisposing factors, namely, hereditary tendencies and abnormal patulency of the nasal cavity." (ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1931.) This latest paper leaves us in the same position.

I beg to present the following hypothesis:

"That atrophic rhinitis is not a disease entity but a syndrome of hypoadrenism, of which the adrenals play an active part, and is only one of many indications of hypoadrenia.

That it is not due to any anatomical condition, as an abnormally patulous nasal chamber. I have never seen a case of atro-

*Read before the American Academy of Ophthalmology and Otolaryngology, September 18, 1931, at French Lick Springs, Indiana.

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phic rhinitis where the turbinates have been removed or in negroes, who have very patent nasal cavities, but who are bountifully supplied by nature with adrenal products.

That any nerve shock to the system, as infectious diseases, childbirth, etc., may so injure the adrenals as to reduce their secretory activity. The loss of the secretory stimulus or hormone on the vegetative sympathetic system may so weaken the heart muscle tone or blood vessel tone as to reduce the flow of blood to such terminal tissues as the skin, hair, mucous membrane and their underlying tissues. To compensate for this weakened terminal circulation, the terminal arteries and veins are reduced in size and atrophy is the result, or reduction of cell stimulus may bring the same end result of atrophy.

The mucous membrane of the nose is most noticeably affected, due to the reduction of the blood supply to the serous glands, causing an aqueous loss in the nasal secretion. The secretion must contain a certain percentage of fluid for physiological functioning of the nasal cavity. As the air passes through the nose, it takes up the aqueous portion but leaves the mucoid portion, thus one of the first nasal symptoms is the presence of a thick, tenacious secretion.

ETIOLOGY.

The underlying cause of atrophic rhinitis is a nutritive disturbance, due to an imbalance of the endocrine glands, particularly the adrenals. This condition is generally launched by a shock to the system, as infectious diseases, focal infections, changes from childhood to adolescence, pregnancy, childbirth, etc. If atrophic rhinitis is already present, any of the above named causes will increase its activity or may cure the case by re-establishing normal endocrine function. One frequently sees cases with atrophy of the turbinated bodies, but a functioning nasal membrane, where the crusts and discharge of early life can be elicited. These generally have compensatory growths in the region of the septum tubercle, uncinat process and the attachment of the middle turbinate. (See "Compensatory Nasal Growths," read before the North Dakota Academy of Otolology, Rhinology and Laryngology, June 1, 1922, by the author.)

Atrophic rhinitis is never present in a hyperendocrine state, but only with a hypoendocrine function. The ozena is a sequela of the condition and is caused by the bacillus fetidus ozenæ or some other fetid germ. One investigator found ozena present 360 times in 1,142 cases of atrophic rhinitis—roughly estimated, about one out of three cases.

If there is excessive secretion, the attenuated mucous membrane of the fossæ and sinuses has been attacked by suppurating germs.

Suppuration and hyperplasia of the accessory sinuses in atrophic rhinitis are secondary and not primary, other than a focal infection.

The condition is not preceded by a hypertrophic rhinitis, and we believe that this theory is based on the fact that there is frequently a hyperplastic suppuration of the ethmoid capsule, which is caused by a secondary infection.

No type of skull seems to predispose to this condition, nor are the negroes, with their wide nasal fossæ, more prone to it.

Surgical removal of the turbinates causes dry nose but not atrophic rhinitis.

HISTOLOGICAL PATHOLOGY.

There is general atrophy of the mucous membrane and its underlying structures, even the turbinated bones. The ciliated epithelium, except islands here and there, is replaced by stratified squamous epithelium. The secretory cells of the mucous membrane of the nose are mixed, mucous and serous glands using a common duct. The glands are distinguished from each other by the fact that the serous glands have secretory capillaries, while the mucous glands do not. The connective tissue left in the underlying structure contracts and first obliterates the secretory capillaries of the serous glands and also interferes with the normal exosmosis of fluid through the membrane, thus taking away nearly all of the aqueous element of the normal nasal secretion.

This atrophy extends to the sinuses, pharynx, larynx and trachea, as well as to the normal lymphoid tissue. The atrophic degenerated state of the sinuses invites infection, thus producing a chronic suppurative sinusitis, accompanied by a purulent discharge.

DIAGNOSIS.

To successfully treat any condition, it must be diagnosed early. There are many signs and symptoms of atrophic rhinitis which appear long before we have the classic crusts, with their sequela of ozena.

The patient comes, either with reference to the crusts that form in the nose, causing obstruction, or because of the disagreeable odor complained of by the family or some friend. They are often unable to discern this odor themselves, owing to the atrophic destruction of the olfactory nerve endings or from pressure of the hyperplastic ethmoid capsule.

The general appearance of the patient shows all the typical signs and symptoms of hypoadrenia: poorly nourished and anemic, with the senile appearance far beyond his years; thin, early graying hair, atrophic skin with its yellowish gray hue and premature wrinkles. They complain of fatigue and lack of vigor and are uninterested in the normal routine of life. The basal metabolism is reduced. The blood pressure is generally below 120. Pulse is slow and weak or rapid and thready.

Feeling along the side of the ridge of the nose where the lateral nasal cartilage joins the nasal bones, one will find atrophic depressions demonstrated by the sensation of touch, which in the later stages, are easily noticed by the eye (Pratt's sign).

On examining the nasal cavity, the membrane will be found pale and the cavity larger than normal. There will be thick, viscid secretion clinging to the membrane and the patient may complain or at least confirm upon questioning that he has considerable viscid discharge which is hard to dislodge. This is due, undoubtedly, to the loss of the watery element of the secretion and the inability of the cilia to move it. There is not an increase of nasal discharge but rather the reverse, unless there has been a secondary infection of the sinuses. This change in the character of the secretion is noticed before any sinus changes by transillumination and X-ray can be demonstrated.

As the condition progresses, with atrophy of the membrane and enlargement of the nasal chamber, the loss of the aqueous portion of the secretion allows it to dry quickly by the passing

air through the nose, and forms a crusty scale on the mucous membrane. In drying, the crusts pull away from the membrane, allowing the heavy mucus underneath to form a second crust. If the crusts are not removed they will eventually fill the nasal cavity. The action of putrefactive bacteria establishes the ozena. Keeping the nose free of crusts will banish the odor, unless the bacteria reach the sinuses. In atrophic rhinitis, when there is no odor, the crusts are dry. However, when odor is present, there is partial liquefaction and heavy mucus is attached to the crusts when they are removed.

The first perceptible structural atrophy usually affects the lower turbinate (due possibly to its blood supply coming from a terminal blood vessel). A secondary infection may cause a hyperplastic ethmoiditis, which will fill the upper half of the nose, giving an atrophic lower turbinate and a seemingly hypertrophied middle turbinate. Polypi are rarely, if ever, present.

When the sinuses are transilluminated and X-rayed in advanced cases of atrophic rhinitis, they invariably show a sinusitis.

DIFFERENTIAL DIAGNOSIS.

With the symptoms, examination and findings as above, there will be no difficulty in diagnosing an atrophic rhinitis. A syphilitic condition can be differentiated by the ulceration and necrosis plus the Wassermann test. In cases of foreign bodies or rhinoliths, the odor is not present after their removal. Tuberculosis of the mucous membrane shows ulcerations. The sinus infections alone are quite distinctive. It must be remembered, however, that there may be an association of these diseases with atrophic rhinitis. It is easy to differentiate the dry noses caused by surgical operations or cauterization.

In the cases of so-called one-sided atrophic rhinitis caused by extreme deflections of the septum, there will be found an attempted compensatory enlargement of the lower turbinate and that the discharge and crusts are caused by sinusitis. We are not dealing with a true atrophic rhinitis.

PROGNOSIS.

All the literature written in the past has given no encouragement in the prognosis. I wish to bring before you the fact, as

stated in our book, "Intranasal Surgery," published in 1925, that the prognosis is good, if the case is properly treated.

TREATMENT.*

The sheet anchor of the treatment is the use of dessicated suprarenal gland (Armour). Two grain tablets are prescribed, to be taken three times a day, preferably at meal time. Only the dessicated gland should be used. Other gland therapy may be combined, if necessary.

If there is an ethmoiditis, the ethmoid capsule should be exenterated, but the middle turbinate must not be disturbed. All ostia of the sinuses should be patulous, and if not, should be made so. Intranasal window resections should be performed upon the antra if necessary, but the lower turbinate must be preserved. An external operation on the sinuses is not indicated.

If hypothyroidism is present, the dessicated thyroid gland, one grain tablet, is given, to be taken three times a day. The patient must be watched carefully to prevent the accumulative effects of the gland. Anemia or any other dyscrasia should receive attention. Hydrargyrum protoiodid tablets, grain one-tenth, given three times a day, stimulate the action of the endocrines.

A nasal oil should be dropped in the nostrils twice a day, the patient being in a supine position.

Packs of ichtholdine (Mulford) or a 3 per cent tincture of iodine in glycerin is indicated, to soften the crusts and stimulate the mucous membrane. Nasal douches of saline solutions are unnecessary.

Improvement is noticed within a very few days, and any odor, if present, will disappear in from one to two weeks, providing the sinuses are not invaded by the ozena germ. The patient will first notice the ease of cleansing the nose of crusts, the improvement in the general well being and in the mental attitude. The mucous membrane will become pink and the secretion will improve towards its normal character, due to the stimulation of the circulation and of the cells.

*The treatment suggested is the one followed by us for the past fifteen years at the University Dispensary, the Minneapolis General Hospital, and in our private practice.

The gland treatment should be continued for some months or until there is a normal secretion of the endocrines, indicated by the disappearance of all symptoms.

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XVII.

CONTINUOUS RHYTHMIC MOVEMENTS OF THE PALATE, PHARYNX, AND LARYNX.

NORMAN LESHIN, M. D.,

CHICAGO.

Continuous rhythmic movements of the palate, pharynx, and larynx occurring synchronously are comparatively rare and of considerable interest because they signify evidence of organic brain disease. In reviewing the literature, Dr. Stone and I¹ were able to find only 27 cases, including our own, showing these movements. In addition to these, Jelinek and Sachs² and Childrey and Parker³ have each reported a case bringing the total number to 29. These cases should not be confused with those having localized movements of the palate which are by far more common and frequently functional in etiology.

The characteristics of these movements are that they involve the palate, pharynx and larynx. They are constant, synchronous, gross and oscillatory, having two phases, a rapid and slow, bearing no relation to the pulse or respiration. They present under anesthesia and are not influenced by extraneous stimulation. The physiologic functions are not interfered with. The movements always occupy the same plane and direction and have a constant rhythm. Their rate is between 120 and 180 per minute, rarely above or below these points. The patients are generally unaware of their presence. There is usually no evidence of local paralysis or disease. The laryngeal movements are generally bilateral, whereas those of the palate and pharynx may be either unilateral or bilateral.

The movements of the soft palate are in a vertical plane. The palate rises and falls with each excursion, elevation being quicker than the depression. Phonation and swallowing do not usually interrupt the spasms. The posterior faucial pillars in a bilateral involvement approach one another in the midline and then return, adduction being quicker than abduction. The rise and fall of the palate give one the impression of a curtain being raised and low-

ered with the pharyngeal aperture becoming higher and narrower in the first phase and then assuming its natural low and wide position in the second. The posterior pharyngeal wall in a bilateral involvement shows no definite movement. In a unilateral disturbance the entire posterior wall, including the naso- and hypo-pharyngeal region, moves toward the affected side and back again. The vocal cords and arytenoids are in continuous motion approaching one another and then separating, occupying usually one-third to two-thirds their normal excursion, never meeting in the midline during the spasms. Adduction is slightly quicker than abduction. The movements on one side may be greater than those on the opposite side. The rate is usually the same and the movements are synchronous with those of the palate and pharynx. The false cords and aryepiglottic areas may also be in synchronous motion. On phonation the cords move normally, stopping the contractions as long as the cords are held in this position; the spasms immediately start as soon as the cords are relaxed. The tension of the cords is unimpaired and sensation is normal. In all the cases except five, in which there was a unilateral movement of the palate and pharynx, both sides of the larynx were affected.

The composite picture presented by these contractions is a quick phase showing the rising of the palate, adduction of both posterior and faucial pillars, vocal cords and arytenoids, followed by a slower movement of these parts in the opposite direction. In the unilateral involvement, the rapid phase consists of the lateral movement of the posterior pharyngeal wall toward the affected side, associated with the medial movement of the posterior faucial pillar and rising of the soft palate on the same side with usually the adduction of both vocal cords and arytenoids. Occasionally there are rhythmic movements of the lower part of the face, the floor of the mouth, neck, aryepiglottic folds and other muscle groups, which may or may not be synchronous with the spasms described.

The isolated movements of the palate have practically the same characteristics as those associated with the pharyngeal and laryngeal contractions. In addition they are usually accompanied by a clicking sound which may be heard when one is close to the

patient and which the patient localizes as being present in the ears. According to Valentin,⁴ these movements follow a nervous or mental shock of some sort in emotional, unstable persons, who are always conscious of them. They usually have periods of remission and cease during sleep. Valentin compared these contractions to a habit spasm which may occur as a result of voluntary repetition in children at an early age, later becoming a habit similar to a mental tic.

Case report: An opportunity to study these movements presented itself in a case recently seen and reported by Dr. Stone and myself.¹ A man, 42 years of age, gave a history of progressive weakness of the left arm and leg for the past five years, requiring at present a cane for support. Two years ago a coarse tremor of the left hand and leg developed. The tremor was made worse by an attempt at movement. For the past year the patient has become more sensitive to cold. His vision has become blurred and reading had been difficult.

The neurologic findings briefly were a left hemiparesis, including the face and tongue. The tongue was coarsely tremulous and protruded to the left. The left upper extremity showed many postplegic hyperkinetic movements; the left lower extremity was markedly spastic and ataxic. The right lower was moderately ataxic. The deep reflexes were bilaterally brisk, the left more than the right. The abdominal and cremasteric reflexes were absent on the left. The Babinski and an exhaustible ankle clonus were present on the left. There was a spontaneous mixed horizontal and rotary nystagmus when looking in either direction and a vertical nystagmus when looking upward.

Examination of the pharynx revealed the entire posterior pharyngeal wall in continuous rhythmic to and fro movements to the left in a horizontal plane. The movements had two components, a slightly quicker movement to the left and a slower one returning. The rate taken repeatedly at various times was always 132 per minute. The rhythm always remained the same, but the tempo would vary occasionally, especially with manipulation of the tongue, when a few quicker movements would interrupt the regular ones. Laughing, swallowing or phonation did not interrupt

the oscillations. The muscles of the posterior wall of the nasopharynx and hypopharynx were similarly involved. The left posterior faucial pillar was also in rhythmic motion, moving toward the midline and back again, synchronously with the movements of the pharyngeal wall and at the same rate. Here also the movements had two phases, the excursion medially being somewhat faster than the returning lateral one. The excursion of the pillar transmitted slight movements of the palate, especially on the left side, which seemed to rise and fall. The uvula, which pointed slightly to the right, showed a rather rotary swinging motion backward and upward. All these movements were synchronous, at the same rate, with slight transient variations in tempo, composed of two components, with no relation to the pulse or respiration. The movement of the pharynx to the left was accompanied by the excursion of the left posterior pillar to the right toward the midline, giving one the impression that the pillar was pulling the pharyngeal wall toward the left around some fixed point. At the same time there was a slight elevation of the palate and the uvula. The return movements were together also, in the opposite direction. The normal movements of the palate and the palatal reflex were not impaired. When elevated the palate deviated slightly to the right. The sensations in the palate and pharynx were not diminished.

Examination of the larynx made indirectly, using the laryngeal mirror, showed both vocal cords and arytenoid in similar rhythmic motion. The cords and arytenoids approached each other and then returned, the movements occupying only approximately one-third their normal excursions, never meeting in the midline during these oscillations. The movements also had two phases, the adduction being quicker than the abduction. The right vocal cord and arytenoid moved more than those on the left. The rate was also 132 per minute, but the regularity seemed to vary more than in the pharynx. The smaller excursions interspersed the regular ones more frequently, and at times those on the left appeared to stop entirely for a brief pause. During the regular tempo, the laryngeal oscillations seemed to be synchronous with those of the pharynx, the adduction of the cords and arytenoids occurring at the same time as the movement of the pharynx to

the left and of the left posterior pillar to the midline. Slight movements were also present in the false cords and both aryepiglottic folds, especially the right. The normal movements of the vocal cords and arytenoids were unimpaired, meeting in the midline during phonation and showing no evidence of paresis or changes in tension. The respiratory movements did not affect the rhythmic oscillations. During phonation the movements stopped and the cords remained quiet as long as they were held in the midline, resuming their rhythmic excursion as soon as they were permitted to open. The movements of the larynx could be felt by placing the finger on the neck in the notch of the thyroid cartilage.

The patient was not conscious of any of these movements. They did not interfere in any way with the physiologic functions of swallowing, speaking and breathing. The movements were always present, even during anesthesia, as was seen when the patient was under ether narcosis for esophagoscopy. The movements of the pharynx, pillar, palate and larynx were exactly the same as when the patient was conscious. An esophagoscopy was done (Dr. S. J. Pearlman) to determine whether the movements involved the muscles of the esophagus. No involvement was present. The nasopharynx was also examined with the nasopharyngoscope. Rhythmic movements were seen on the left lateral wall, referred from the left posterior pillar. There was a suggestion of a slight movement of the outer lip of the eustachian tube, with no opening and closing contractions at the opening of the tube. The right lateral wall of the nasopharynx showed no movements.

A diagnosis of multiple sclerosis was made.

Spencer⁵ reported the first case showing these synchronous movements in 1886. Oppenheim,⁶ in 1889, reported two cases, one of epidemic cerebrospinal meningitis and the second of a cerebellar tumor found at postmortem examination. Five years later Scheinman⁷ reported the first case of unilateral movements of the larynx associated with unilateral involvement of the palate and pharynx in a case of syphilis of the brain. Within the next few years Uckerman,⁸ Bernhardt,⁹ Semon,¹⁰ Steward¹¹ and Pegler¹² each reported a case. In 1904, Klien¹³ published two cases in complete detail with postmortem findings. Both

revealed cerebral as well as cerebellar disease. Sinnhuber¹⁴ also reported a case the same year, and Semon¹⁵ a case of possible tumor of the pons a year later. In 1909, Porter¹⁶ reported the second case of unilateral laryngeal movements synchronous with those of the palate and pharynx. Graeffner¹⁷ published two cases in 1910, one revealing a large lesion in the right cerebral hemisphere and many smaller lesions in the pons, brain stem and cerebellum on postmortem examination. He also cited a case of Leipmann's,¹⁸ Klien,¹⁹ in 1918, reported two more cases, making four in all, that he had seen. The first revealed many hemorrhagic areas in both the cerebrum and cerebellum on postmortem examination. The second case was one of arteriosclerosis. The same year Kelly²⁰ published a postmortem report of a case showing arteriosclerosis and softening with a hemorrhage into the cerebellum and capillary fibrosis particularly marked in the cerebellum. Pfeiffer²¹ the following year reported these movements in a case following a gunshot wound in the left occiput with injury to the underlying brain. Bloch and Lemoine²² reported a case of cerebellar disease with pseudobulbar paralysis in 1926. In 1928, Freystadt²³ reported three cases with a complete description of these movements and their characteristics. Wilson,²⁴ the same year, and Jelinek and Sachs,² and Childrey and Parker³ recently each reported cases showing unilateral laryngeal movements with unilateral palatal and pharyngeal involvement.

Twenty-four of the twenty-seven cases reviewed¹ showed definite evidence of organic brain disease. The three cases not included (Bernhardt,⁹ Semon,¹⁰ Pegler¹²) had insufficient reports to make a neurologic diagnosis. The subsequent two cases mentioned^{2,3} also revealed definite disease of the central nervous system. Nineteen cases in our series revealed positive evidence of cerebellar dysfunction, fourteen had findings of cerebral arteriosclerosis and three sufficient clinical evidence to make a diagnosis of bulbar palsy. Only six cases included postmortem reports. All these revealed cerebellar as well as cerebral pathology. In three of these reported by Klien, left sided movements were associated with left cerebellar disease, right sided movements with right cerebellar pathology and bilateral movements with bilateral cerebellar involvement. In view of the great majority of cases

surveyed revealing organic disease of the central nervous system, one seems justified in assuming the presence of organic brain disease when these characteristic synchronous rhythmic movements are present. However, due to the small number of post-mortem examinations accompanying the cases and the varied pathology found, one cannot as yet ascribe any definite localizing significance to them.

Schultzen,²⁵ Baginsky,²⁶ Boeck²⁷ and Ernst²⁸ each report a case of hysteria having spasms of a similar nature. These spasms were much less in frequency, ranging between 50 to 70 per minute, and in one case synchronous with the pulse and another with the respirations. No evidence of organic disease was present in any of the cases. The movements found, associated with hysteria, should offer no difficulty in differential diagnosis. In this condition the movements are usually localized to the palate, larynx or pharynx. They are not constantly regular but tend to be irregular and disappear when the patient's attention is drawn to something else or while he is asleep. The rate is usually slower than that found in the organic cases, less than 120 per minute, and may be synchronous with the pulse or respiration. The patient is usually aware of these movements and seeks medical attention for that purpose. Voluntary effort will usually stop the spasm. Frequently those of the palate and pharynx are associated with an audible click. There are no objective evidences of organic disease of the central nervous system, but other signs of neurosis, such as hysterical anesthesia, hyperesthesia and paralysis, may be present.

SUMMARY.

Continuous synchronous rhythmic movements of the palate, pharynx and larynx are comparatively rare; only twenty-nine cases have been found in the literature.

The review of these cases shows a definite relationship of these movements to organic disease of the central nervous system.

No localizing neurologic significance can be ascribed to them as yet because of the varied pathology found in the few post-mortem examinations made.

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XVIII.

TREATMENT OF PAPILLOMAS OF THE LARYNX WITH CALCINED MAGNESIA.*

BENJAMIN KATZ, M. D.,

LOS ANGELES.

The persistent tendency of laryngeal papillomas to recur after removal sometimes incapacitates the patient for life (R. Abbe¹) or even results in his death (Bruns,² Rosenberg,³ H. Todd,⁴ Shallcross and Bayley,⁵ S. Jones,⁶ H. Smith,⁷ Wm. Duffy⁸). Numerous methods of treatment have been tried in an effort to combat their recurrences. The merits of these methods can be seen only in the proper perspective, if a study of the end results, available from case reports and accumulated statistical material is made; and if the feasibility of the application of the method, in regard to the anatomic relationships of the affected organ, is taken into consideration.

The methods of treatment can be classified as follows:

1. Operative, extra- and endo-laryngeal.
2. X-ray and radium.
3. Electrocauterization and fulguration.
4. Therapeutic methods.
5. Combinations of methods.

OPERATIVE METHODS.

Extralaryngeal.—In the operative methods seniority belongs to the extralaryngeal method, as the endolaryngeal path of operation became possible only after Czermak introduced the laryngeal mirror (1859).

Tracheotomy has the first place in extralaryngeal operation as the oldest surgical method and also the most frequently applied.

The next step in extralaryngeal surgery was made by Brauer, who in one case opened the thyroid cartilage for the removal of the papillomas.

*Read at a meeting of Los Angeles Society of Ophthalmology and Otolaryngology, October 12, 1931.

Bruns introduced the endolaryngeal way of removing the papillomas and since then a discussion has arisen over the merits of these two methods. To bring the discussion to the realm of facts Bruns made a statistical study of 54 cases of thyrotomy in papillomas of the larynx and of 40 cases of endolaryngeal removal of the growths. These statistics show that there is a recurrence after thyrotomy in 42 per cent, functional defects in 63 per cent, deaths in children of $9\frac{1}{2}$ per cent. In the endolaryngeal group there were no lethal results, recurrence in $12\frac{1}{2}$ per cent, functional defect in 32 per cent. Bruns prefers the endolaryngeal indirect method to the external operations.

A. Rosenberg continued Bruns' statistics, collecting and analyzing 88 thyrotomies in children and 48 endolaryngeal operations. In the series treated by thyrotomy, recurrences were in 38 per cent, functional defects in 15 per cent, deaths in 19 per cent, while in the group of endolaryngeal operations recurrences were encountered in 10 per cent and deaths in 6 per cent.

Comparing statistical data in both Bruns' and Rosenberg's series, it is clearly evident that the results in the endolaryngeal group are far more favorable than in the one treated by thyrotomy.

What advantages are claimed for thyrotomy by enthusiasts of this method over the endolaryngeal route? Navratil⁹ and Masini,¹⁰ in the beginning of the discussion, stated that it is possible to accomplish a radical cure by a single operation.

Another argument in favor of thyrotomy is that the endolaryngeal method in multiple papillomas of small children can be carried out only with difficulty.

Statistics of Bruns and Rosenberg show, however, that about half of the cases treated by thyrotomy have relapses and the operation has to be repeated two or more times. Rosenberg cites a case in which thyrotomy was performed seventeen times on one child.

By analyzing the age of the patients, it is possible to prove that even in multiple papillomas of small children the endolaryngeal method can be carried out successfully, because in cases treated in this way the children suffered not only from aphonia but also from dyspnea as a result of multiple growths.

Another proof that very early age is not a contraindication to the endolaryngeal method can be seen from the fact that nine children treated by this method were under four years of age and in about 50 per cent of them there was complete recovery.

The space in the larynx of small children is so limited that access to the affected part during thyrotomy is insufficient to admit even a finger. Not all parts of the larynx can be easily inspected, particularly the more remote ones, such as the aryepiglottic ligaments or the laryngeal surface of the epiglottis. Because of the limited space, the possibility of removing the entire growth radically is very problematic; the energetic efforts to remove all the growth in a single operation have the result that the original simple stenosis due to papillomas is replaced by a more dangerous and permanent stenosis due to scar formation. (Thost.¹¹)

In addition to not having accomplished the desired results, thyrotomy often causes considerable functional damage to the delicate vocal organ of a growing child. The thyroid cartilage, the main support of the larynx in general, the most important part of attachment of the vocal cords particularly, is split in thyrotomy. Even in the case of immediate reunion of both parts, there cannot be obtained a perfect and solid fusion. In the most successful cases both lamina of the thyroid cartilage unite, but the natural firmness is never restored. In unfortunate instances the fusion is very feeble and occasionally dislocation and excessive motility of both laminae of the thyroid cartilage can be seen a long time after operation. Sometimes, in the line of division, granulation tissue and excessive scar formation develop. Therefore, it is possible that the cords become separated at their point of attachment, with consequent disturbance of their function. In small children, that can happen very easily since the anterior commissura does not make a sharp angle, but rather a semicircle, and therefore exact division in the middle line is hardly possible.

In spite of all the above disadvantages of thyrotomy, this operation is performed for papillomas of the larynx on children at the present time.

All the criticism about thyrotomy can be, in a greater degree, applied to thyrostomy, in which the larynx is kept open for

months and even years. This operation for papillomas was first performed by Ruggi in Italy, but in detail was worked out by Sargnon and Barlatier. It is true that when the larynx is kept open it is possible to remove all new growth methodically and systematically. While in single instances good results were obtained, in others very grave complications developed.

At the Belgian congress in 1910, M. Beco¹³ reported three cases of thyrostomy for papillomas of the larynx. In the first case the larynx remained open for nine months, in the second for one year and eight months, and in the third the stoma was kept open for two years. This operation makes a helpless invalid of the patient, causing loss of voice and consequent grave functional damage to the larynx. Therefore, it should be condemned for use in papillomas.

B. Endolaryngeal Operations, Indirect Method.—The endolaryngeal removal of papillomas by indirect method is usually performed under local anesthesia, using the spoon-shaped laryngeal forceps. Rosenberg, Shambaugh¹⁴ and a few others use the snare. The advantage of this instrument is that the field of operation is not obstructed, but special care should be taken to prevent the removed pieces from slipping into the bronchi. Lichtwitz uses a window tube, puts the growth into the window and then removes the papillomas in the tube by sharp instruments or snare. Lori proposes a metal catheter, No. 7 to 10, with oval openings on the side and sharp cutting edges.

Direct Method.—The endolaryngeal removal of papillomas can be performed also by the direct method. One year after Kirstein published his method of direct laryngoscopy, Rosenberg employed this method for removing papillomas in children, performing the operation under chloroform anesthesia.

That this method is sometimes carried to extreme limits can be seen from the case of Sir St. Clair Thomson, who at the meeting of the London Laryngological Section of the Royal Society presented a boy, six years old, cured by direct laryngoscopy after the boy had been submitted to operations under chloroform anesthesia eighteen times. It is possible to perform this method on children under one year of age (Ganalejo¹⁵), using a cocaine-adrenalin anesthesia (Garel¹⁶), or without anesthesia (McKin-

ney,¹⁷ R. Johnston¹⁸), V. Schmigelow,¹⁹ Albrecht,²⁰ Hubbard and Galbraith²¹ favor suspended laryngoscopy under general anesthesia. Andre, Moulonguet, Pierquin and Richard²² remove the growths under suspended laryngoscopy and then apply radium.

It was noticed by many authors that the juices of the neoplasm may cause transplantation of growth. Ullman²³ proved this experimentally. He inoculated himself in the arm with a mixture of papillomas from a child's larynx by the method of Pirquet and injected the material into the mucous membrane of a dog. Three months later a mulberry-like mass developed on his arm and papillomas in the dog. Experiment with the filtrable virus gave also positive results. Therefore, all manipulations with the tubular spatula in the execution of the direct method should be very gentle. New formation of growths on previously healthy parts of the larynx was noticed by investigators as a result of pressure or impingement by the tube. (Hubbart,²⁴ Smith,⁷ Hubbard and Galbraith²¹).

RADIUM AND X-RAY.

The new method of treating papillomas of the larynx by radium, reported in 1911 at the international congress in Berlin by L. Polyak,²⁵ aroused great hopes. Polyak introduced radium into the larynx in two cases of papillomas, using a cylindrical aluminum capsule 4 mm. in diameter with walls 0.5 mm. thick. The capsule was attached to a laryngeal sound and introduced into the larynx under cocain anesthesia and injection of morphin. The patient was able to keep the capsule in the larynx for one-half to two and one-half hours. In the first case irradiation was performed for 20 hours and 54 minutes during fifteen seances; in the second case for ten hours in six seances. According to Polyak, the growth disappeared, the voice became loud and strong. R. Abbe employed a different technic: he performed a preliminary tracheotomy, introduced a wire through the wound into the mouth, attached to it a capsule with 100 mgr. of radium and pulled the capsule into the larynx so that it remained between the vocal cords for thirty minutes. Three months later there was no sign of papillomas. Some use a rubber tube or bougie for lodging the radium, introducing them from above (Smith, Iglauer) or

through the tracheal wound (S. Jones, Wm. Duffy). The time of application usually varies from one-half to two and one-half hours. Mainsin and Wildenberg²⁶ used a special technic. They introduced into the larynx in two cases 25 mg. of radium enclosed into a specially constructed sound. The dose of radium is spread out in a series of tubes in order to obtain a uniform irradiation. Simultaneously radium is applied from the outside. Their cases were not completed at publication. Guisez recommends that the radium should be carefully filtered, because a quick reaction can follow. He advises always to perform a preliminary tracheotomy on account of the possibility of an acute edema of the larynx.

The proper position of the capsule in the larynx is controlled usually by a laryngeal mirror. P. Hickey places the patient before the fluoroscope for this purpose.

Results of Radium Treatments.—Very early it became evident that radium does not guarantee a cure nor insure against recurrences in cases declared to be cured. Killian had already reported unsuccessful cases which he thought resulted from insufficient doses of radium. However, larger doses up to 100 mg., applied by later authors in longer exposures, do not prevent recurrences (Jones, Hickey, Smith). Complications, such as cicatricial stenosis necessitating an emergency tracheotomy, developed (F. Hopkins²⁷). In one case the radium tube was applied continually for twenty-eight hours (S. Jones). Even such prolonged application did not insure against recurrence, which developed three months later. In spite of repeated irradiations the papillomas continued to grow. The child died suddenly from suffocation.

In the case of Duffy, of the Johns Hopkins University, in which radium was applied, death followed the development of necrotizing laryngitis and tracheitis, tracheo-esophageal fistula and a diffuse degeneration of the thyroid gland, although the dose (100 mg.) and exposure (one and one-half hours) were of the usual character.

X-Ray Treatments.—Attempts to treat papillomas of the larynx with X-ray were even less encouraging than with radium. Case reports of this method are not very numerous. P. Hickey reports that he gave a patient "a thorough course of X-ray treatments but no beneficial effect was noted."

In the case of Drs. Shallcross and Bayley the patient twice developed severe burns from roentgen ray treatments, but no results were accomplished and the authors discontinued the raying. S. Jones, in one case, gave twenty-four seances of X-ray irradiation to a woman twenty-five years of age. The papillomata underwent no recession. "It would seem," says Jones, "that little is to be hoped from either radium or X-ray treatments in papillomas of the larynx."

ELECTROCAUTERIZATION AND FULGURATION.

In single instances electrocauterization has been used. Abbey* reported a case in which thyrotomy was performed four times with recurrence of the growth after each operation, although the bases of the tumor were cauterized. In the case of Hickey, recurrence took place in a few weeks with the return of so much obstruction that an emergency tracheotomy had to be performed.

Fulguration is used more frequently. The technic described in detail by Smith⁷ and Galbraith,²¹ is very complicated and dangerous, if performed under general anesthesia, because a possibility of explosion is present.

Does the use of this method pay for the efforts which are necessary to carry it out? An analysis of results of treatments is very discouraging: "Fulguration has been disappointing as a permanent and positive relief for these growths in children" (H. Smith). Considerable edema of such threatening character developed in certain cases after fulguration that tracheotomy was necessary. Recurrences take place just as often as in other methods (Harris and Culbert, Duffy). As a result of fulguration scar formation in the larynx develops and phonation is impaired (Hubbard and Galbraith).

THERAPEUTIC MEDICATION.

Concerning the therapeutic treatments some authors, especially English, as Korner,²⁸ Koellrentter²⁹ use arsenic. Building up of the general health is important in undernourished children, because microscopic examination of papillomas in some instances revealed the presence of T. B. bacilli (Steiner³⁰). In certain cases

*Smith, Harmon: Papilloma of the Larynx. J. A. M. A., Vol. LXIII, No. 25, Dec. 19, 1914, p. 2204.

of papillomas, improvement occurred from trichloroacetic acid, which is a very good remedy in laryngeal tuberculosis (B. Katz³¹). It is possible that the underlying nature of the growths in these cases was of a tuberculous character. The list of medicamentations tried in papillomas is endless. Alcoholic solution of salicylic acid (D. Grant), spray from solution of formalin, 1/2000 to 1/250 (Bronner), chromic acid (Baumgarten), lactic acid (Tovolyi), Thuja (Shurly) and many other therapeutic agents.

TREATMENTS WITH CALCINED MAGNESIA.

In the same year when radium was introduced for the treatment of papillomas, Claoué³² published his observations about treatment of laryngeal papillomas with calcined magnesia. Dr. Frech used this remedy with very good results in cutaneous warts, which are histologically similar to papillomas, and by his advice Dr. Claoué decided to try this remedy in the latter. Calcined magnesia is used also with good results in veterinary surgery, especially for papillomas in the mouths of dogs. Claoué had under his observation two children, five and six years of age, with aphonia and multiple papillomas, who had recurrences after previous surgical removal of the growths. In these two cases Claoué started treatments with calcined magnesia, half gram three times a day, and in two weeks there was a remarkable improvement of the voice and clearing up of the larynx. Besides his own two cases, Claoué cited a case treated in the same way by Sargnon, the author of thyrostomy. Schiffrers³³ in one instance gave calcined magnesia to a woman, forty years of age, for hyperacidity of the stomach who coincidentally suffered from recurrent papillomas of the larynx. It was noticed that recurrences ceased. After Claoué's paper a few other authors published their observations with this method. Durtu³⁴ reported two cases with good results. He pointed out that it required about six months to obtain a cure in his cases, one of which was a child, three and one-half years old, the other a man, 56 years of age. In both cases the growths were removed previously by surgery without success. S. Masini,³⁵ the former defender of thyrotomy, published one case of a woman who had papillomas removed many times during a period of 25 years and finally developed stenosis, for which tra-

cheotomy was performed. After three months of treatment with calcined magnesia the papillomas completely disappeared.

There are a few reports of unsuccessful results with this method. Falgar³⁶ reported two cases and Broeckaert³⁷ one. During discussion Beco remarked that a six weeks' period of treatment in the last case is not sufficient for final results. The case published by Luiggi Leto³⁸ demonstrates that lack of success in treatments with calcined magnesia may depend upon insufficient dose. Leto ordered 4.0 to 6.0 of this remedy daily to a patient 19 years old, who had recurrences after operative removal. When after forty days of treatment there was no improvement, he increased the daily dose to 8.0. Following this dose the papillomas began to regress and finally disappeared.

As far as the action of calcined magnesia in papillomas is concerned, Prof. Slosse and Dr. Reding believe that this remedy re-establishes the deficient cellular metabolism by adjusting the equilibrium of the mineral elements of the cell.

PERSONAL OBSERVATIONS.

The writer used calcined magnesia in three cases of papillomas of the larynx with remarkably good results, having published the case reports previously.³⁹ Because in all cases aphonia was associated with dyspnea, as a result of multiple papillomas, the growths were removed from the very beginning by the indirect endolaryngeal method, using sharp spoon-shaped forceps, under local cocain-adrenalin anesthesia. Simultaneously with surgical removal of papillomas, the patients have been treated with calcined magnesia, one-half to one gram, three times a day. Two of the patients were children, ten to eleven years of age, and both stood the cocainization with 10 per cent solution very well. The indirect endolaryngeal removal could be carried out without difficulty after the children were trained in a few previous seances to endolaryngeal manipulations.

I would like to mention briefly the essentials of these case reports:

Case 1.—E. D., ten years old. Aphonia and dyspnea. Multiple papillomas on both vocal cords and in interarytenoid space. Had tonsillitis often, after one attack of which voice became hoarse and gradually difficulty in breathing developed; an emergency thyrotomy was performed, papillomas

removed and wound closed. After temporary improvement, recurrences with difficulty in breathing developed. A thyrotomy was done again but papillomas continued to recur. Under cocain-adrenalin anesthesia the main parts of growth were removed in a few seances by indirect endolaryngeal technique and calcined magnesia three times daily ordered. One month later the larynx was free from papillomas. Two months after admission the patient left the hospital.

Case 2.—Catherine G., 19 years of age, was admitted with hoarseness and difficulty in breathing. Diphtheria at 10. At 14 had a cold after which she lost her voice. Gradually difficulty in breathing developed for which she was admitted to the hospital. General health normal; syphilis, tuberculosis absent. Multiple papillomas on vocal cords and in intra-arytenoid space. Aphonia, dyspnea. By indirect method the growth was removed in a few seances under local anesthesia. The patient, for the sake of observation, intentionally was kept for a month without magnesia; the papillomas continued to grow. Then the growths were removed again and calcined magnesia ordered. Two months from the beginning of the treatments with magnesia, no trace of papillomas was present. After an additional month of observation, the patient was discharged.

Case 3.—Girl, 11 years of age. Admitted with hoarseness and difficulty in breathing. Measles at 7, following which the voice became hoarse and dyspnea gradually developed. General condition good. Tuberculosis, syphilis absent. Larynx: multiple papillomas on true and false vocal cords. Papillomas removed by indirect method in five seances under local anesthesia, after previously training the child to laryngeal manipulations. Calcined magnesia internally. Five weeks later was free from papillomas, voice became louder, no recurrences. Patient discharged with advice to continue calcined magnesia.

COMMENT.

Summing up the facts about different methods of treatment of papillomas of the larynx, it should be noted, first, that among surgical external operations, it is permissible to employ the tracheotomy in vital indications in general and in multiple papillomas of small children particularly.

Thyrotomy and thyrostomy result in anatomic and functional damage to the organ, do not insure against recurrences and do not give any advantages for removal of the growth. Therefore, these operations for papillomas of the larynx should be completely discarded.

X-ray proved to be of no value in laryngeal papillomas and should not be applied.

While in single instances good results were reported from radium, in the majority of cases no cure was accomplished and recurrences appear after temporary improvement. Complications,

such as edema, scar formation, necrosis of healthy parts of the larynx, degeneration of the thyroid gland with consequent death, were reported. The new technic of using a strong filter and a series of small tubes to spread out the action of radium was applied only in a single instance, and the results were not completed at the time of publication. The dose of radium (25 mg.) used in this technic is not sufficient for accomplishing definite results, as was already reported by Killian in his case. The enthusiastic expectations that radium would be specific in papillomas were not fulfilled after practical experimentation.

Electrocauterization and fulguration do not stop the growth of papillomas, do not prevent relapses, may cause an acute edema and result in permanent stenosis of the larynx. There is danger of explosion if general anesthesia is employed. Both the patient and the physician are better off if these methods are not used.

In very small children with multiple papillomas it is preferable to perform a preliminary tracheotomy and then to remove the growth through the tracheal opening or through the larynx by indirect or direct technic. The indirect endolaryngeal method can be carried out even in children after preliminary training to laryngeal manipulations. If the direct method is used, great care should be exercised not to injure the healthy parts of the larynx with the spatula, because practically and experimentally it has been proven that the virus of the growth is transferable.

In cases of dyspnea, when the larynx itself is free from the growth, bronchoscopy should be performed through a tracheal wound because deaths from asphyxia have been reported, in which, during postmortem examination, papillomatous growths were found under the tracheal tube and over the bifurcation of the bronchus.

While others employed calcined magnesia alone for treating papillomas, the writer, to shorten the time of treatments, combined calcined magnesia with endolaryngeal indirect removal of the main parts of the growths, the results obtained being highly satisfactory.

Calcined magnesia should be given for a period of three to four months in increased doses, from 4.0 to 8.0 daily

A short interval of rest of from one to two weeks should be given after this period and then calcined magnesia continued again for another three to four months in smaller doses.

There are no facts to affirm that calcined magnesia is a specific remedy for laryngeal papillomas or that it is able to bring about their spontaneous retrogression in all instances. However, the experience of different investigators demonstrates that it diminishes the energy of the growths and the chances of recurrence, probably by re-establishing the normal metabolism, particularly by adjusting the equilibrium of the mineral elements of the body cells.

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XIX.

ELECTROSURGICAL TONSILLECTOMY: ADVANTAGES AND LIMITATIONS OF THE FRACTIONAL ELECTROCOAGULATION METHOD.

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Although surgery probably will continue to be the method of choice for the removal of tonsils in the large majority of cases, radiation therapy and diathermy for special cases merit consideration, in view of recent advances in these fields. Knox¹ experimented with roentgen irradiation of the tonsils and pointed out the indications and limitations of this method. Scal,² Williams³ and Lane⁴ have described the uses of radium for tonsillar hypertrophies. These authors believe that radium treatment is an adequate substitute for tonsillectomy when surgery is, for some reason, contraindicated. Portman,⁵ Dillinger,⁶ Silvers,⁷ Skillern⁸ and others have attempted to evaluate the diathermic removal of tonsils. While roentgen irradiation and radium therapy still claim their adherents, they are not so commonly employed as diathermy. The latter method is now receiving considerable attention because it has been suggested as a substitute for surgery. The main purpose of this paper is to call attention to the fallacies as well as to the possibilities of electrosurgical tonsillectomy.

METHODS IN CURRENT USE.

Among the surgical diathermy methods employed for the removal of tonsils is the one-stage operation. Theoretically the one-stage operation is good, but practically it has proved unsatisfactory. If the removal of the tonsils in one sitting is contemplated, surgery is preferable to any substitute method. When diathermy is employed, it is invariably necessary to repeat the process of destruction for remaining pieces. Furthermore, the local reaction produced by extensive coagulation is often much more severe than that of surgical removal. The fractional or mul-

tiple stage method is the only rational one. There need be no definite number of treatments. The operation is continued at successive sittings until all the tonsillar tissue is finally removed. A minimum of local reaction results from fractional treatments. The one-stage operation is employed chiefly by those who seek to substitute diathermy for surgery in all cases without regard for specific indications.

Nervous individuals are poor subjects for diathermic extirpation. They do not easily tolerate manipulation in the pharynx. The preparation and anesthesia for each treatment consume as much time as that required for surgical removal. If the case at hand warrants extra inconvenience, and surgical removal cannot be safely performed, these drawbacks need not be seriously entertained. When surgical enucleation is not contraindicated, and particularly when the patient has no choice in the matter, surgery should be employed. The factors already mentioned should at once exclude diathermy, which in some patients causes nearly the same discomfort at each of eight or ten sittings as is required at the one sitting for surgical removal.

During the past six years I have performed over 200 tonsillectomies by electrocoagulation. When an equal number of my surgical removals are compared with those done by electrosurgery, certain definite observations stand out:

1. For local surgical tonsillectomy infiltration of novocain gave an adequately painless field for operation. Infiltration anesthesia proved unsatisfactory for the electrosurgical procedure because the waterlogging of the tissues added greatly to the severity of the local reaction. Topical application of cocain and other solutions was not sufficient to insure against pain from the electrocoagulation treatment. When topical anesthesia was employed, the severe reaction which followed infiltration anesthesia was absent.

2. The local reaction of surgical removal was in most instances less than that of electrosurgery when infiltration of novocain was employed. The local reaction from electrosurgery was always less with topical anesthesia than surgery with infiltration.

3. The period of convalescence from surgical tonsillectomy varied from two to ten days, while the average time of absence

from occupational duties was three days. When fractional electrocoagulation was used, the patients attended their usual duties, and in only a few instances were unable to partake of their regular meals.

4. Hospitalization was routine in the surgical removals, while the office served for the electrosurgical method.

5. In the surgical tonsillectomies no secondary hemorrhages were encountered, and in only one case was there a severe hemorrhage at the time of operation. No primary or secondary hemorrhages occurred in my series of electrosurgical removals.

6. There were no unusual complications from either the surgical or the electrosurgical methods.

INDICATIONS FOR ELECTROSURGICAL TONSILLECTOMY.

The question finally resolves itself: "Are there any absolute indications for removing tonsils by electrosurgery in preference to surgery?"

An outstanding indication is hemophilia. The physician hesitates to remove the tonsils of a patient with true hemophilia or of one who has delayed clotting time of the blood from some other cause. This applies, also, but to a lesser degree, to cardiac patients. While surgery can be performed successfully in a large percentage of the latter, a substitute method is more desirable, especially so if surgical shock can be avoided. In my hands electrocoagulation of the tonsils has been successfully carried out in more than forty patients with all types of cardiac lesions.

The removal of the tonsils in the tuberculous without unfavorably influencing the pulmonary process has always been a serious problem. Recent investigations by the author⁹ have demonstrated that diathermy is a reasonably safe method for extirpating the tonsils in the tuberculous.

Marked hypertension is not definitely a contraindication to surgical tonsillectomy. Should complicating factors make it so, fractional diathermic extirpation can be carried out with greater safety.

One of the accepted indications for electrocoagulation is the destruction of tonsillar stumps. The author¹⁰ has called attention

to the use of electrocoagulation for the destruction of recurrent lymphoid tissue in or about the tonsillar bed after tonsillectomy.

DISADVANTAGES OF THE ELECTROSURGICAL METHOD.

In spite of the growing enthusiasm for this newer means of removing tonsils, there are numerous disadvantages. One is the special study required to learn the technic, due to the apparent simplicity of the procedure. Physicians may attempt it without adequate training and experience and secure bad results. Diathermy, incorrectly employed, can produce the same mutilation of normal structures as poor surgery. Frecker¹¹ struck the keynote of this situation when he stated: "Electrocoagulation is an operation to be used only in very expert hands and then only with great caution and strict limitation of the current and its effects to the tonsillar tissue."

A further disadvantage is the lack of uniformity of technic. With the more general utilization of electrosurgery each worker has developed and advocated his own special technic. The result has been a disagreement on the part of specialists in correctly evaluating the method as a whole. Trainor¹² points out that all those who use electrocoagulation are of the opinion that the results achieved are satisfactory, no matter what technic they may employ. This is far from true, as there are any number of physicians who have tried electrocoagulation and have discarded it because of poor results. The failures probably were due to the types of electrodes employed, the attempt to remove all the tonsil at one sitting and the use of infiltration anesthesia. Some specialists have since re-adopted electrocoagulation because they have observed favorable results which have followed the adoption of improved electrodes, fractional technic and topical anesthesia. The disadvantage due to a lack of uniformity of technic may eventually be overcome by the adoption of a standard method.

ADVANTAGES OF THE ELECTROSURGICAL METHOD.

Prominent among the advantages is the fact that patients exhibit little or no fear for the method. Numerous individuals whose tonsils have long ago been condemned, but who have hesitated because of fear of surgery and hospitalization, have promptly submitted to electrocoagulation.

With electrosurgical removal of the tonsils there is little pain on swallowing and thus no difficulty to eat regular meals. While this is of no special significance in the normal individual, it is important to some groups of patients, such as the tuberculous, in whom the loss of a few meals may temporarily retard favorable progress of the systemic disease.

We have been told that electrosurgical removal of the tonsils is a bloodless method. It would be more accurate to say that it is nearly always a bloodless method. This is a decided advantage when one analyzes the potential dangers of abnormal bleeding in some surgical tonsillectomies. The chief danger in electrosurgery is secondary hemorrhage. This may occur in from two days to ten or twelve days after a treatment. The cause is sometimes an aberrant vessel which breaks through the coagulated tissue or the use of too strong a current and the inclusion of too much tissue at a single treatment.

The prevention of shock is always an important consideration. The manipulation entailed in an electrocoagulation treatment does not produce shock. The procedure can be performed without the slightest fear of causing a systemic upset, even in extremely delicate individuals.

CONCLUSIONS.

1. While diathermy for removing the tonsils has gained popular favor and has become the method of choice of some specialists, its use is not to be recommended as a substitute for surgery for the majority of cases.

2. There are, however, definite advantages in an adequate substitute method when surgery is considered hazardous in the presence of certain systemic diseases.

3. Since electrocoagulation of the tonsils is a scientifically sound method for selected cases, the specialist should familiarize himself with its technic.

30 NORTH MICHIGAN AVENUE.

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XX.

THE BÁRÁNY NOISE APPARATUS.*

B. M. BECKER, M. D.,

BROOKLYN.

Heretofore the Bárány noise apparatus has been utilized for two well known purposes, namely, (1) to deafen one ear temporarily while the other is being subjected to functional hearing tests by air conduction, and (2) to assist in the detection of malingering, in the Lombard test. To these may be added (3) as an aid in functional hearing tests by bone conduction and (4) as an aid in unmasking pretended deafness by bone conduction tests.

1. In testing the auditory function of a patient it is essential, in order to determine the hearing capacity of each ear separately, to eliminate one ear from the process while the other is being examined. The best means, to date, of accomplishing it, is by means of the Bárány noise apparatus. This instrument owes its efficacy partly to occlusion of the ear canal but chiefly to its noise which so overwhelms the perceptive mechanism of the ear that for all practical purposes it may be regarded as absolutely sound-proof. The only exceptions are the Galton whistle and the screaming voice, at the ear.

2. Lombard's test consists in inserting the nipple of the Bárány instrument into the sound ear of the patient and setting it in operation while he is instructed to read from a printed page. In a person simulating deafness the voice will not be raised at all or only to a very slight extent; whereas in one who suffers total deafness in the unobstructed ear the voice will be raised considerably, even to actual screaming, when subjected to the test.

3. In considering bone conduction tests it is found that they are subject to errors, not only because of their inherent imperfections, but also because of our inability to rule out one ear while

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testing the other as is done in air conduction tests. In all these tests the opposite ear must of necessity participate, in a lesser or greater degree, thus injecting an element of uncertainty in the result of the examination.

For instance, a patient presents himself complaining of deafness in the right ear. By air conduction tests we find him totally deaf in the right ear. On applying a vibrating fork to the vertex (Weber's test) he localizes its sound either in the vicinity of the fork or in the left ear, and on placing the fork over the right mastoid he is not certain where he hears the sound or lateralizes it to the left. Can we logically conclude from all these tests that the left ear is the sole seat of his auditory perceptions? How can we, with absolute certainty, maintain that the right cochlea is totally functionless? These questions could be answered with perfect assurance only when the left labyrinth could be completely eliminated when examining the right.

The writer devised a method, described elsewhere,¹ whereby a current of air directed against the drum membranes will prevent all sounds, both by air and bone conduction, from reaching the perceptive mechanism. However, considering the difficulties attending this method he, by further experimentation, with a new Bárány noise apparatus (having used previously an old instrument, with negative findings), found it to answer the purpose admirably. All sounds by bone conduction are absolutely eliminated from the ear in which the instrument is used. The patient is rendered totally deaf, on that side, to both air borne and bone conducted sounds, save for the exceptions noted above.

That this instrument, when used in the usual manner, produces total temporary perceptive deafness can also be deduced from a consideration of the principles underlying Lombard's test.

One hears the sound of his own voice by air and bone conduction. It is for this reason that a patient suffering from a unilateral conductive lesion will often hear his own voice louder in the affected ear than in the sound one. Furthermore, when both ears are thus affected he will often complain of hearing his own voice louder than usual. This condition can be duplicated by a simple experiment. Close one ear with the palm of the hand pressed, not too strongly, against it and breathe deeply. The breath sounds

will become lateralized in the occluded ear. Whisper, feebly, the sounds of the letters "k," "p" and "q" or of similar labials and palatals and they likewise will be lateralized to the obstructed ear. Now, with the ears open, pronounce, inaudibly, some syllables or words; close both ears in the manner described and the sounds become audible, remove your hands from the ears and they again become imperceptible—the tone of voice remaining the same.

From the above described experiments it is readily seen that bone conduction plays a considerable rôle in the hearing of our own voices and hearing the voice is necessary to its regulation. Had the noise apparatus in Lombard's test produced a condition akin to conductive deafness the patient would hear his own voice the louder and the test would thereby become nullified. It is only because the apparatus causes almost complete perceptive deafness that he raises his voice as a compensatory measure.

4. This test depends upon two factors. The one is that one cochlea can be completely ruled out by bone conduction while testing its mate, and the other is that one cannot readily localize sounds conveyed through the skull.

The test consists in placing a vibrating fork (128) on the vertex and at each mastoid process successively while the Bárány apparatus is held in the right ear and then the same process is repeated with the apparatus in the left ear. His responses to the questions whether he hears the fork sounds or not and where he hears them, marked in simple symbols, constitute the evidence upon which a diagnosis is based. Thus: a plus sign (+) signifies that he hears the sound; a minus sign (—) that he does not hear it. If he hears it in the right ear (+R) is used; if in the left (+L); and if he cannot localize the sound it is signified by (\pm).

Example: We are confronted with a patient who complains of deafness in the right ear. On physical examination we obtain negative findings. All tests by air conduction give negative results. The sound of the fork when applied to the vertex and to each mastoid is referred to the left. Here we are in the presence of a patient who is a sufferer of a grave perceptive lesion or a malingerer. We next proceed with the Bárány apparatus in his right ear. With the fork on the vertex the sufferer will state that he hears the sound near the fork and in some cases he will

refer it to the left ear. The malingerer, anxious to emphasize the good condition of his left ear, will refer the sound to the left. When the fork is placed next on the right mastoid the sufferer will state that he hears the sound but he cannot decide where or he will refer it to the left; the malingerer will state that he hears no sound or he hears it in the left ear. The reason for his first answer is that fearing exposure he will deny hearing any sound unless he is certain of its being perceived by the left ear, and this, as stated previously, cannot readily be determined in bone conducted sounds. On the other hand, he may decide beforehand, in order to be consistent, to refer all sounds to the left. With the fork on the left mastoid he will refer the sound to the left.

The instrument is now placed in the left ear and the same procedure followed. With the fork on the vertex, the sufferer from a total perceptive lesion, will give a negative answer. For, his right ear being deaf by virtue of his lesion and the left ear being deafened by the apparatus in it, he will thus be totally deaf, binaurally. For the same reason his answers will likewise be negative when the fork is applied to each mastoid process. The malingerer, however, for the reasons stated before, is likely to refer the fork-sound, from each of the three regions, to the left ear, and thus become exposed.

This test, while simple to the examiner, is quite confusing to the malingerer and the most astute and coached simulator is bound to ensnare himself during one stage or another of the test either by his direct answers or by inconsistencies in his responses when the test is repeated.

For the sake of comparison and ready reference a tabulated chart is herewith appended representing four types of individuals subjected to this test with their most likely responses to it.

- (A) One with the left ear normal and the right normal.
- (B) One with left ear normal and a conductive lesion in the right.
- (C) One with left ear normal and perceptive lesion in right.
- (D) One with left ear normal and simulated deafness in right (malingerer).

With the Bárány apparatus in right ear:

Fork on vertex	Fork on right mastoid	Fork on left mastoid
(A) \pm	(A) \pm	(A) + L
(B) \pm	(B) \pm	(B) + L
(C) \pm	(C) \pm	(C) + L
(D) + L	(D) - R + L	(D) + L

With the Bárány apparatus in left ear:

(A) \pm	(A) + R	(A) \pm
(B) + R	(B) + R	(B) + R
(C) -	(C) -	(C) -
(D) + L	(D) + L	(D) + L

By comparing the responses of the patient with the schematic tabulation outlined above, one can at a glance detect any discrepancies between the two and draw his conclusions therefrom.

The above is part of a scheme, by the same author, which appeared in another publication.²

4802 EIGHTEENTH AVE.

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XXI.

LATERAL SINUS THROMBOSIS: A REVIEW OF 184 CASES WITH SPECIAL REFERENCE TO THE POSTOPERATIVE TYPE.

LAWRENCE R. BOIES, M. D.,*

MINNEAPOLIS.

Brunner¹ reported statistics on lateral sinus thrombosis developing in the postoperative period and emphasizes the high mortality in this event. In the twenty-two cases which he records, 50 per cent ended fatally.

His conception of a true postoperative thrombosis entails the absence of any history or operative findings suggestive of the condition at the time of primary operation and the development of complicating signs after a convalescent interval, usually two weeks or more, followed by demonstration of the thrombosis at the subsequent investigation of the mastoid cavity. The invasion of the lateral sinus takes place, therefore, entirely within the postoperative period.

Another time relationship in the occurrence of diagnostic signs of lateral sinus involvement after operation is discussed by Brunner¹ under the term "latent" thrombosis. This applies to those cases in which insufficient symptoms or signs are present at time of operation to justify a diagnosis, but the condition becomes manifest early postoperatively.

If one applies this classification, lateral sinus thrombosis may be: (1) present at the time of primary operation on the mastoid, with evidence of its existence; (2) latent, in that it becomes manifest because of the operation, or (3) postoperative, developing entirely in the period after operation.

In the latent group, it is assumed that a thrombosis has taken place, but the signs of it are not manifest, and that a continued

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thrombus formation and the production of symptoms occurs as a result of the "stirring up" due to operation. The first two groups above mentioned are not distinctly separable as to event but only as to manifestation.

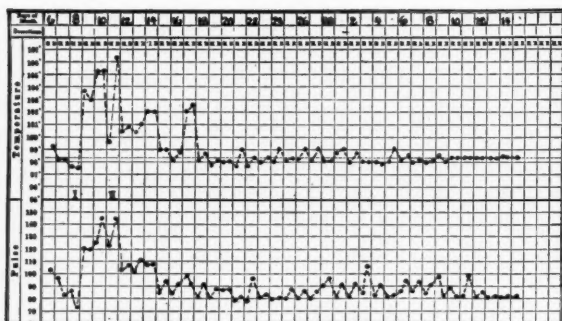
My interest in this study was stimulated by Brunner's report. This paper is based on the records of lateral sinus thrombosis at the Massachusetts Eye and Ear Infirmary during the past fifteen years. The purpose of this study of the records was to find out how often postoperative thrombosis of the lateral sinus occurred in this hospital, and to determine the amount of success in the treatment of this condition. Several case summaries are presented to illustrate the latent and postoperative types of thrombosis. For brevity, all laboratory tests, consultants' reports and other details not of positive information are omitted in these summaries:

CASE I—A LATENT TYPE.

I. On February 8 a simple mastoidectomy was performed on an 18-year-girl because of a profuse, purulent discharge increasing in amount three weeks after the onset, with X-ray evidence of destruction in the mastoid. There was no history of significant fever, sweats, chills, headache, nausea or vomiting. At operation, a pneumatic mastoid in coalescent state was thoroughly cleaned of pus and granulations; all apparent cells were excoriated. The sinus was not exposed.

On the following day, her temperature rose to 103.6 degrees, she complained of headache, had several chills, was nauseated and vomited.

CHART I.



There was a recurrence of the chills on February 10. A blood culture taken on the previous day was reported to contain a growth of a streptococcus hemolyticus.

II. The mastoid cavity was investigated again with exposure of a perisinus abscess on removal of the sinus plate. The sinus was then opened, revealing an obturating thrombus, with no bleeding from below. The thrombus was removed and jugular ligation performed.

Three subsequent blood cultures were positive for a streptococcus hemolyticus and a fourth was negative. Transfusion was given. An abscess developed at the site of a hypodermic injection in a deltoid muscle and had to be drained. Convalescence was otherwise uneventful.

Comment.—The absence of any signs or symptoms which would suggest a diagnosis of thrombosis at the time of operation is obvious. The extensive thrombus formation found so soon after mastoidectomy indicates that it existed prior to operation.

CASE II—POSTOPERATIVE TYPE.

I. A 14-year-old boy was operated on July 13 because of postaural swelling, tenderness and fluctuation in the course of a profusely discharging ear which showed a sagging of the posterior superior canal wall. There was no history of headaches, chills, sweats, nausea or vomiting. This was an acute exacerbation in the course of a chronic otitis. X-ray examination had indicated an area of destruction over the lateral sinus in a mastoid otherwise sclerosed. These findings were substantiated at operation; the sinus was found with an area of healthy appearing granulations overlying. The sinus was then uncovered more widely and appeared to be normal.

Healing was slow but the convalescence otherwise uneventful for two and one-half weeks following the operation.

II. On August 6, because of a continued postaural discharge and the temperature manifestation as indicated on the chart, the mastoid cavity was investigated. It was filled with granulations. A few small tip cells were exenterated; otherwise nothing significant was found.

Again the wound failed to heal and the patient seemed unable to make a satisfactory convalescence. No enlightening information followed the usual laboratory examination of blood, the wound secretions, etc. No significant general physical findings were suggested by a medical consultant.

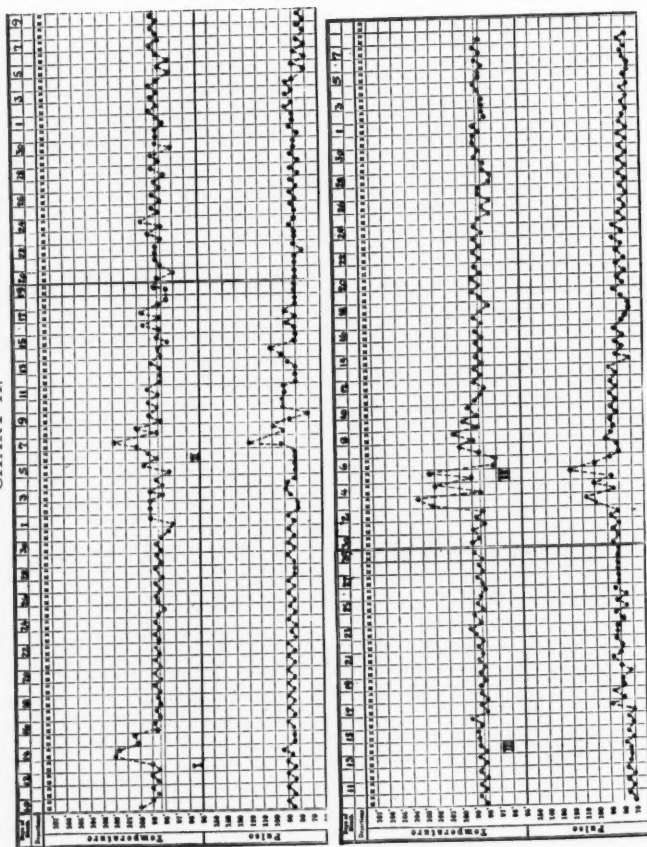
III. On September 14 a radical mastoidectomy was done. The sinus was widely uncovered and some dura exposed above the tegmen of the mastoid. There were no significant findings, and no indication of the involvement of the lateral sinus. Healing was more satisfactory following this operation but again was slow.

A sudden rise in temperature and in pulse rate occurred on October 3. A diagnosis of lateral sinus thrombosis was made within the next twenty-four hours.

IV. Jugular ligation was performed and on opening the sinus, a thrombus partially filling the lumen was found. This was removed and convalescence was uneventful from this date.

Comment.—The evidence in this case that thrombosis developed in the postoperative period rests on the following:

CHART II.



1. Absence of any suggestive symptoms prior to the first operation.
2. Onset of symptoms pointing toward lateral sinus invasion two and one-half months after the primary operation.
3. On three occasions examination of the lateral sinus gave no indication of thrombus formation within.
4. The incomplete thrombosis found on incision of the lateral sinus.

The findings at primary operation suggest a setting for invasion of the lateral sinus, that is, a condition of exposure brought on by necrosis. This, however, is not an uncommon finding in mastoids with chronic pathology in a sclerosed structure. It occurs without subsequent signs of sinus pathology ever developing. On the other hand, the postoperative condition of an exposed lateral sinus, undoubtedly bathed in pus over a considerable period of time, must be considered favorable to invasion of the sinus.

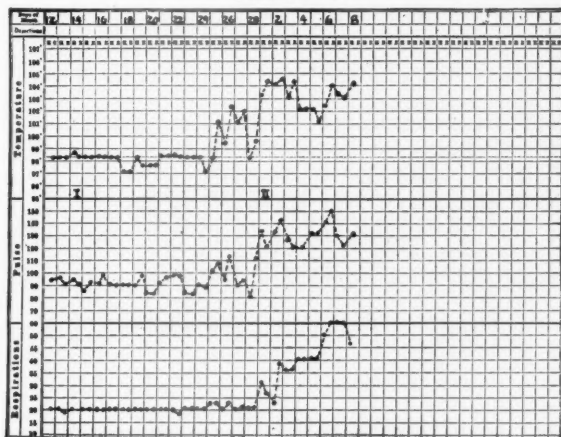
Speculation as to the cause of slow wound healing after the first, second and third operative attacks on the mastoid cannot offer any definite evidence as to why the wound failed to heal promptly. Involved are the factors of resistance and the organism at fault.

It seems probable that the actual invasion of the sinus began after the radical operation.

CASE III—POSTOPERATIVE TYPE.

A simple mastoidectomy was done on a 15-year-old boy on February 14 because of a profuse purulent discharge from an ear which had been draining for three or four weeks, and from which the patient was experiencing intermittent pain and headache in the right parietal area. There was tenderness over the mastoid tip on admission; his temperature was normal and he gave no history of, sweats, chills, fever, nausea or vomiting. An X-ray examination indicated destruction in the mastoid tip.

CHART III.



I. At operation there was no evidence of anything to suggest lateral sinus involvement, but the plate was not removed as it appeared to be healthy. The pneumatic structure of the mastoid was filled with pus and the partitions were partially broken down.

Convalescence was rapid with prompt healing of the postaural wound; the middle ear was dry by February 19 and the patient was allowed to be up and about the ward.

On February 24 he complained of pain in his ear again and the middle ear began to discharge. There was a sudden rise of temperature on the following day; the subsequent course is graphically illustrated on the chart.

II. On March 1 the internal jugular vein was ligated and the sinus investigated, revealing a necrotic thrombus with frank pus in the sinus. A blood culture had grown a streptococcus. Pneumonia developed, the patient became jaundiced, complained of pain in one wrist and developed liver tenderness. Death occurred on March 8. No autopsy was obtained.

Comment.—A ten day interval existed between the time of primary operation at which a rather ordinary form of mastoiditis was seen, and the development of acute symptoms suggestive of lateral sinus invasion. As the chart indicates, there was nothing manifested by the temperature or pulse to suggest anything abnormal in this period. Though the sinus plate was not removed at the primary operation, it is reasonable to believe on the evidence at hand that no sinus pathology existed, though it is a matter of common experience that extensive pathology may exist under a healthy looking sinus plate.

CASE IV—POSTOPERATIVE TYPE.

I. A 10-year-old boy had a simple mastoidectomy on December 10 on an ear with a history of suppuration for one month, with sudden cessation of discharge five days before admission, followed by earache again and postaural tenderness and swelling. He gave a history of three or four short chills in the two days previous to admission, and said that he had been feverish for the past week. There had been no sweats, headaches, nausea or vomiting.

At operation perforation of the cortex was found and considerable pneumatization in the zygomatic region with suppuration most marked at this site. The sinus plate appeared to be healthy. All apparent cells were exenterated.

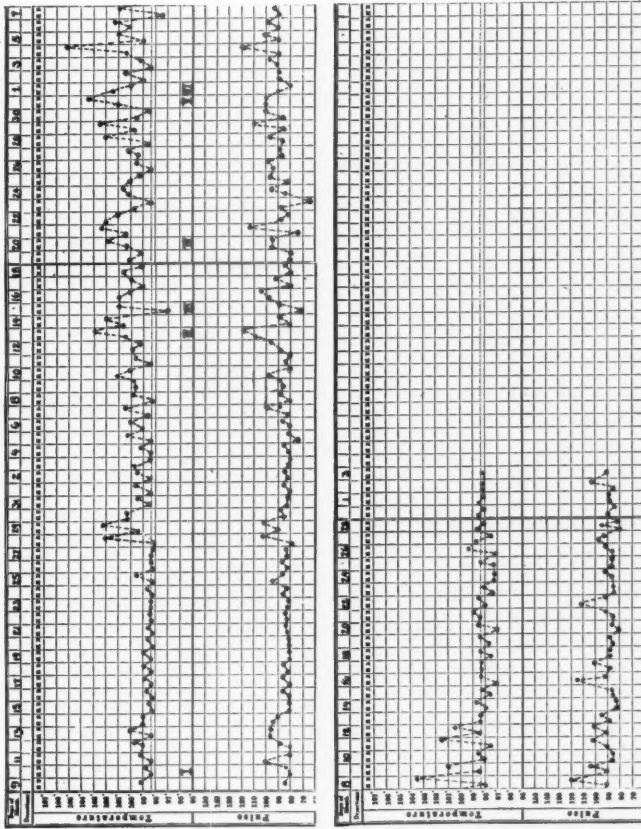
During the following eighteen days convalescence was uneventful but the wound healed slowly. On December 28 the postaural wound began to discharge and the patient complained of some parietal headache. His temperature rose to 102 degrees, and in the succeeding six weeks underwent the fluctuation shown by the chart.

II. The mastoid was reopened on January 13, revealing a cavity containing pus and granulations. All cells apparently had been exenterated.

III. On January 15 a right lateral rectus paralysis developed; the patient still complained of headache. A blood culture taken two days previously was reported to contain a growth of a staphylococcus; this was considered a contamination.

On January 16 the wound was again investigated and the lateral sinus uncovered, revealing no evidence of disease.

CHART IV.



IV. A Tobey-Ayer test was negative on January 20. The spinal fluid gave a cell count of 125 lymphocytes but was otherwise negative for any significant findings.

On January 24 a transfusion of whole blood was given. The red cell count had been 2,840,000 with fifty per cent hemoglobin on January 20. A neurological service consultant reported no findings additional to those already mentioned.

V. A Tobey-Ayer test was positive on January 31; the spinal fluid cell count was 602, all lymphocytes, and otherwise negative.

VI. Jugular ligation was performed on February 1; the lateral sinus was found to be completely thrombosed. As much of the clot as could be was removed, but no bleeding was obtained from either end. Convalescence was uneventful from this date.

Comment.—The history of three or four short chills in the two days previous to admission might point toward a lateral sinus invasion. A dependable statement as to the patient's febrile condition was not available.

The finding of a normal appearing lateral sinus at secondary operation over a month after the primary operation indicates, however that the lateral sinus had up to that time not been involved. Supporting the development of a thrombosis after that date are also the two Tobey-Ayer tests, one negative on January 20 and the positive one eleven days later.

The temperature fluctuations and the discharge from December 28 through the succeeding month can be accounted for by the evident supuration deep within the petrous process, producing in this case the elements of the clinical picture of a Gradenigo syndrome or a "petrositis".

In the following Table I, summary is made of the incidence of these types of thrombosis, the mortality percentages, and the factor of an acute versus a chronic otitis in 184 cases occurring at the Massachusetts Eye and Ear Infirmary since 1915. In all, 202 cases are recorded in this period, but 18 of them were admitted in a condition beyond hope of surgical aid, and are therefore excluded from this analysis. Brunner's statistics on total mortality rate and postoperative thrombosis are inserted in the table for comparison.

DISCUSSION.

It is evident from this report that the mortality from postoperative sinus thrombosis in this series of cases, as compared with Brunner's, is much lower (15.7 per cent and 50 per cent). Brunner suggests no reason for the high mortality rate. As causal factors in the development of a postoperative thrombosis, he feels that the virulence of the organism, stagnation of secretions and a sinus wall well forward and near the cortex play an important part.

TABLE I.

	No. of Cases	No. of Deaths	Mortality Percent
Thrombosis manifest at time of operation	120	34	28.3
Latent thrombosis	45	9	20.3
Postoperative thrombosis	19	3	15.7
(Brunner's cases of postoperative thrombosis)	22	11	50.0
Total, Massachusetts Eye and Ear Infirmary, 1915-1930	184	46	25.0
(Brunner's cases, total)	152	33	22.2
Due to an acute otitis:			
1. Manifest at operation	71	17	23.9
2. Latent	37	8	21.6
3. Postoperative	18	3	16.6
Total	126	28	22.2
Due to a chronic otitis:			
1. Manifest at operation	49	7	34.7
2. Latent	8	1	12.5
3. Postoperative	1
Total	58	18	31.0

In the majority of the cases in our series, where thrombosis developed after operation, the secondary exploration of the mastoid wound revealed suppuration with a considerable accumulation of secretion. Obviously, a virulent organism is a prominent factor. The operative records state that in several of the cases a forward sinus near the cortex was found.

From a speculative standpoint, one can think of few reasons why the invasion of the lateral sinus in the postoperative period, especially after a considerable convalescent interval, should carry a greater hazard than in the latent condition, or one in which evidence of the thrombosis is present when the case first comes to the surgeon's care. The offending organism is probably the same in the lateral sinus infection as has been the prominent one in the mastoid disease, the "walling off" process has had an opportunity in the postoperative interval, and the host has had some chance to develop an immunity and perhaps gain some ground in a convalescent way from the effects of the suppurative process. These factors might well be expected to work toward a lesser risk in postoperative sinus thrombosis.

Explanation of the marked variation in the mortality rates for these two groups must be a matter of speculation. Brunner's

series is collected from cases, some of which date back previous to 1908. The method of mastoid operation has undergone changes since that period, especially in some American clinics. In Brunner's case reports, the operation in some instances is described as an antrotomy. In practically all of the 184 cases in the Infirmary group, the operations consisted of an anatomic exenteration of all existing cells. It is possible that an incompletely exenterated mastoid with continued suppuration in the presence of virulent organisms might, in the presence of postoperative invasion of the lateral sinus, carry a greater hazard than in a mastoid completely cleaned out at primary operation.

Exposure of the sinus at operation is not a fundamental factor, Brunner agrees, because this is a procedure too frequently performed in comparison to the number of cases of postoperative thrombosis that occur.

COMPLICATIONS.

The incidence of complications in the group of 184 cases amounted to 43 per cent—that is, in nearly one-half. Cases in which a diagnosis of septicemia was made are not included in this number. Many of the cases had positive blood cultures; others had clinical evidence of septicemia in which blood culture taken previous to the operation on the sinus was negative. In a sense, I assume that in all cases of lateral sinus disease, as manifested by the symptoms of fever, sweats and chills, a diagnosis of septicemia applies in some period of the illness.

Table II lists the chief complications and the mortality in each group. Pulmonary complications in order of frequency included pneumonia, septic infarcts, pleurisy, empyema and embolus. The orthopedic conditions were chiefly septic joints. The cases of brain abscess were temporosphenoidal or cerebellar in location. The one case of erysipelas which ended fatally was severely ill with a septicemia on admission. In a number of instances more than one condition existed, but only the major illness is included in this enumeration.

The relation of the number of complications to the special type of thrombosis shows too little variation between types to be of significance. Thus, 7 of the 19 cases of postoperative thrombosis developed complications (36.8 per cent), 18 of the 45 cases

of the latent type (40 per cent), and in the group in which thrombosis was evident at operation, 56 of the 120 cases (46.6 per cent). In this group last mentioned, as previously emphasized, are included many cases in which there had been delay in getting medical care, complication existed on admission, and the risk was definitely greater.

TABLE II.

Complication	No. of Cases	No. of Deaths	Mortality Percent
Meningitis	12	12	100.
Cavernous sinus thrombosis.....	3	3	100.
Pulmonary	27	17	62.9
Brain abscess	7	3	42.8
Orthopedic	11	2	18.1
Erysipelas	7	1	14.2
Other	12	2	16.6
Total	79	40	50.6

SUMMARY.

A study of the records of 184 cases of lateral sinus thrombosis treated at the Massachusetts Eye and Ear Infirmary between 1915 and 1930 indicates that:

1. The mortality in cases of thrombosis developing in the postoperative period is lower than in the latent type or in the type in which thrombosis is evident at the time of primary operation for mastoiditis. In 19 cases of postoperative thrombosis there were fatalities in 3 cases (15.7 per cent), as compared to 11 deaths in 22 cases (50 per cent), as recorded by Brunner.

2. The total mortality in all cases of lateral sinus thrombosis was 25 per cent.

3. The mortality is higher in thrombosis occurring in chronic suppurations than in the acute.

4. The incidence of all types of thrombosis is greater in acute suppurations.

5. The incidence of complications is high (46.6 per cent). Pulmonary complications are the most frequent, with intracranial and orthopedic conditions next in order.

1833 MEDICAL ARTS BLDG.

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XXII.

CONGENITAL BILATERAL ATRESIA OF THE NASOPHARYNX: REPORT OF A CASE.

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In reviewing literature of congenital obstruction of the nasopharynx one finds little definite material. Stewart¹ reports two cases of bilateral and four cases of unilateral congenital stenosis.

Because of the apparent rarity of the condition, I wish to report one case that came under my observation recently.

The reports above mentioned were of females, as was the case which I had. This was a baby, weighing six pounds. She was five hours old when I first saw her, and her father, a physician, was giving artificial respiration and oxygen. The child was cyanotic and struggling for breath. The child had extreme difficulty in respiration from birth.

After cleaning out a considerable amount of thick, glairy mucus from the nose and shrinking the tissues, one could see a white glistening membrane, apparently closing both sides of the nose. Using a cotton tipped probe to sound the obstruction, it was found that a complete bony wall was occluding both openings into the pharynx.

The head was too small to permit passing the finger behind the soft palate, so a bent probe was used to palpate the posterior surface of the obstruction. Here one could also determine bilateral bony walls.

The oropharynx was small and the root of the tongue had a tendency to go upward and meet the soft palate. This shut off the mouth breathing and accounted for the cyanosis and difficult breathing.

On account of the small external nares, I used only a small curved antrum trocar to make an opening on each side. On the left side was a thin plate of bone through which the instrument passed quite easily into the nasopharynx. The right side, how-

ever, had a double bony wall, which enclosed a relatively triangular shaped space; the bone was thicker and more dense than the left. As soon as these openings were made, the breathing became normal and the color pink. Catheters were inserted in the openings to maintain patency and left in place for twenty-four hours.

The child nursed normally and did well for three weeks, when she began to have difficulty again. On examination, I found both openings practically closed. To establish patency this time I used a small antrum trephine and punched out a small button of bone on each side. There was a better and freer opening on both sides this time. The catheters were reinserted for twenty-four hours. Since then there has been no trouble and the child is thriving.

NIXON BUILDING.

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XXIII.

INTRA-ORAL INJURIES AND INFECTIONS OF THE PHARYNGEAL LOBE OF THE PAROTID GLAND.*

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The parotid gland has several lobes besides the large facial lobe: one runs backward between the sternomastoid and the digastric muscle; a glenoid lobe lies in the posterior part of the glenoid cavity; a pharyngeal lobe runs forward and inward between the styloid process and the external carotid artery towards the pharynx; a pterygoid lobe may also be present and runs forward between the two pterygoid muscles. When the gland is carefully removed without disturbing the surrounding parts, the recess which it occupies is seen to be of a considerable space, between the ramus of the jaw in front and the sternomastoid muscle behind, with a floor formed by two sloping walls, an anterior and a posterior, which meet at an angle, corresponding closely to the styloid process. The fascia covering the parotid is derived from the deep cervical fascia, which divides below to enclose the gland. Traversing the substance of the gland are the temporomaxillary vein, branches of the facial nerve and the external carotid artery, which divides into its two terminal branches in the gland.

The pharyngomaxillary fossa, as described by Mosher,¹ "is more of a potential than an actual space until it is filled with pus. In the baby at birth it is easily demonstrable because it is filled with fat and stands out clearly. In the adult it may be considered as a cone, the base of which is attached to the base of the skull around the jugular foramen. The apex of the cone is at the hyoid bone. Reinforcing the fossa on the outside is the parotid fossa; below is the submaxillary fossa. The carotid sheath emerges

*Read before the Colorado Congress of Ophthalmology and Otolaryngology, Denver, Colorado, July 24, 1931.

from the apex of the cone and for practical purposes continues the fossa or cone through the neck into the thorax.

The inner boundary of the fossa is the superior constrictor with the tonsil attached to it. The outer boundary below is the internal pterygoid muscle lining the inner surface of the ascending ramus of the jaw and mating the masseter on the outside. The outer boundary above is the parotid gland, the gland at this point not being covered with fascia. Posteriorly the prevertebral muscles and the prevertebral fascia bound the fossa. The fossa is divided into unequal parts by the styloid process and the muscles arising from it. These are in front of the great vessels and protect them. On the posterior wall near the middle line on the front face of the second cervical vertebra there are two lymphatic glands which drain the nose and the upper pharynx and are in chain with the deep cervical glands."

The pharyngeal lobe of the parotid gland in its anatomic relationship to the pharyngomaxillary fossæ is variable. The apex of the pharyngeal lobe may extend inward between the two pterygoid muscles, towards the constrictor muscle of the pharynx; and it then becomes a factor in intra-oral surgery, where the muscle may be deficient or may have been perforated in some surgical procedure, or an abnormality of the gland is found where the lobe extends through into the pharyngomaxillary fossæ.

Among the peculiar accidents to which children are prone while playing, I think the following case is sufficiently rare to be recorded:

A mother brought her five-year-old daughter to the hospital with the following history: While running with a stick in her mouth she fell. This stick was about twelve inches long and one-fourth of an inch in diameter. The tip had a ninety-degree angle about one-fourth of an inch from the end, and was shaped like a fish hook. This curved fish hook end of the stick entered the left side of the soft palate internal to the mandible just above the tonsil, with the curved tip pointing upward and outward. She became frightened and pulled the stick out, bringing with it the pharyngeal lobe of the parotid. On the way to the hospital there was a profuse hemorrhage which had practically subsided at examination. Traction on the mass could not be felt through the facial parotid, due to the fact that the pharyngeal parotid is devoid of fascia. That the child was able to protrude, retract and move laterally the inferior maxillary bone ruled out a pterygoid muscle hernia. I replaced this parotid mass which was the size and shape of the thumb into the sphenomaxillary fossæ, and sutured the soft palate with catgut; primary union took place,

and the wound was entirely healed within four days. There was no swelling of the pharyngomaxillary fossa, or of the facial parotid, and no fistula developed.

Infections of the pharyngeal lobe of the parotid gland can enter through the tonsils; through caries of vertebræ; osteomyelitis of the inferior maxillary; by irrigation of purulent maxillary sinusitis where the needle punctures the posterior wall of the sinus carrying the infection into the sphenomaxillary fossa; a faulty technic in an alcohol injection for trifacial neuralgia; following extraction of the third molar, where undue amount of traumatism was produced, or where the molar was lost in the sphenomaxillary fossa; or by penetration of bacteria through the capillary spaces.

Infections of the sphenomaxillary fossa are not uncommon in our practice.

Col. T., sixty years of age, in August, 1930, complained of a mild sore throat of two days duration. Suddenly he had a feeling of fullness on both sides; this was succeeded by difficulty in breathing, a chill, with a temperature of 102.6, tachycardia, leukocytosis of 29,000, polymorphonuclears 92 per cent and albuminuria. The general picture was alarming. The lips were blue, face bathed in sweat, with anxious expression. Deep cervical cellulitis extended to the clavicles on both sides. On intra-oral examination, I found the uvula was edematous; the whole pharynx and lateral pharynx was soft and waterlogged. A number of puncture wounds were made through the soft palate with a cataract knife. For the following forty-eight hours he was irrational and the outcome looked unfavorable. A small amount of seropurulent secretions drained through the puncture wounds. As the drainage became established through the pharyngomaxillary fossa, the deep cervical cellulitis gradually disappeared and recovery took place.

George McClellan² reports: "In several cases in the author's experience with parotid gland infection, where the pharyngeal lobe abscess was so large that it was feared the evacuation of the pus through direct incision by the mouth might suffocate, by its entering the glottis, external drainage was established by careful dissection down to stylomaxillary ligament and tapping the space above referred to just below the lower border of the parotid gland."

CONCLUSIONS.

1. The pharyngeal lobe of the parotid gland can be injured through a nose and throat operation and by intra-oral traumatism.

2. The pharyngeal lobe is devoid of fascia, and susceptible to infection through the pharyngomaxillary fossa with a resultant cervical cellulitis.

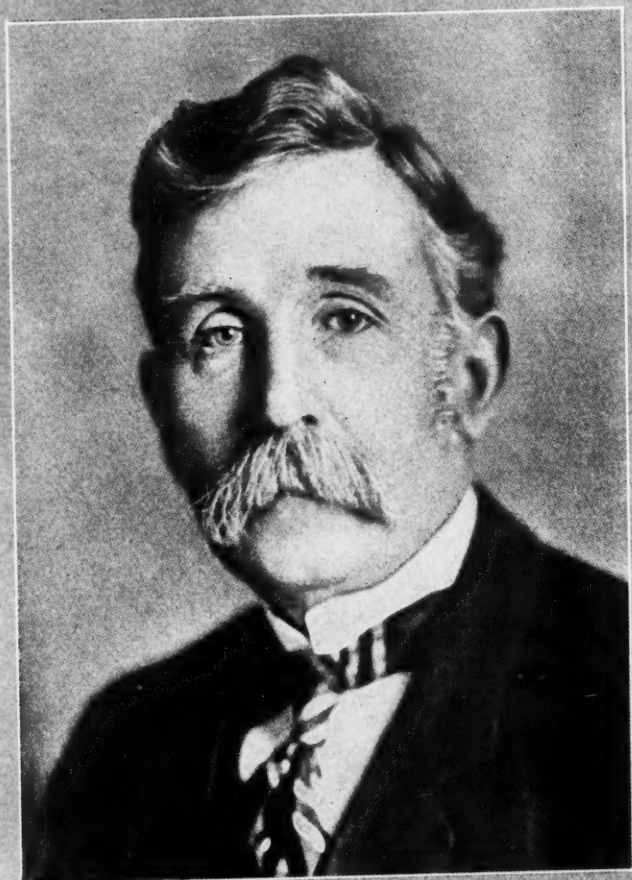
3. Multiple incisions or punctures through the soft palate may afford sufficient drainage; if not, it should be opened by external incision.

4. When recognized and proper surgical measures are pursued, the prognosis is favorable.

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BURTON ALEXANDER RANDALL

BURTON ALEXANDER RANDALL

Burton Alexander Randall, distinguished otologist, died at his home in Philadelphia on January 4, 1932. At the time of his death, Doctor Randall was Emeritus Professor of Otology in the University of Pennsylvania. He had not been in active practice for five or six years, but he continued his affiliations and maintained an active interest in the otological organizations to which he had rendered a long life of service.

He was graduated from the University of Pennsylvania in 1880, later receiving a Ph.D. degree from the same institution. He began his career as demonstrator of histology in the University of Pennsylvania in 1880, following which he held the position of Eye and Ear Surgeon to the Episcopal Hospital from 1882 to 1891 and to the Children's Hospital in 1885. He was Professor of Diseases of the Ear at the Philadelphia Polyclinic from 1888 to 1902, Clinical Professor and Professor of Otology in the University of Pennsylvania from 1891 to 1924. He also held staff appointments in the Polyclinic, Methodist, Children's, Episcopal, Women's and St. Timothy's hospitals.

Doctor Randall was a Fellow of the College of Physicians of Philadelphia, of the American Ophthalmological Society, the American Otological Society, of which he was a former president; the American Laryngological Association and the Philadelphia Pathological Society.

The following list of his contributions to medical literature gives some idea of his activity and versatility. He was for many years a central figure in the otological affairs of his city and of the national organizations to which he belonged. He is survived by his widow, a sister, two brothers, two sons and a daughter.

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JOHN E. MacKENTY.

Dr. John E. MacKenty, senior surgeon of the Manhattan Eye, Ear and Throat Hospital, died unexpectedly December 11, 1931, of angina pectoris, at his home, 111 East Sixty-first street. He was 63 years old.

Connected for thirty-one years with the Manhattan Eye, Ear and Throat Hospital, Dr. MacKenty was one of the pioneers in the development of the major surgery of the sinuses and for cancer of the throat.

With technologists of the Bell Laboratories, he developed an artificial larynx for patients who had suffered removal of the larynx from cancer or other causes. In 1927 his device and method became nationally known because of the prominence given his restoration of the voice of the late Senator T. Coleman du Pont of Delaware.

Dr. MacKenty was born in Richmond, Quebec, in 1869, and was graduated in 1892 from McGill University in Montreal. He went to New York in 1900 and became attached to the staff of Manhattan Eye, Ear and Throat Hospital. His promotion on the staff of that hospital was rapid, and during the last nineteen years he has held the posts of surgeon director in the throat division and senior surgeon of the hospital.

He was a member of the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, New York Academy of Medicine, New York County Medical Society, Medical Society of the State of New York and American Medical Association. He was also a fellow of the American College of Surgeons.

Surviving are his wife and two children, John Gilman MacKenty, connected with the Radio Corporation of America, and Mrs. Wilhelmus B. Bryan, Jr.

PUBLICATIONS OF JOHN E. MacKENTY.

Congenital Occlusion of the Choanæ. *The Medical Record*, published September 7, 1907.

Headache Caused by Pathologic Conditions of the Nose and Its Accessory Sinuses. *Archives of Otology*, Vol. XXXVII, No. 1, published 1908.

Trichinosis of the Upper Respiratory Passages, With Report of Cases. American Medicine, new series, Vol. III, No. 2, pp 69-71, published February, 1908.

The Submucous Operation on the Nasal Septum, With a Plea for a More Rapid Technic. American Journal of Surgery, published May, 1908.

The Radical Frontal Sinus Operations, Type of Operation Best Adapted to the Cure of Sinus Disease With the Least External Deformity. The Medical Record, published July 1, 1911.

Three New Plastic Operations on the Nose and Throat. The Medical Record, November 25, 1911.

Tumors of the Carotid Body. Annals of Surgery for December, 1913.

Operative Treatment of Cleft Palate. ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, published March, 1915.

Surgical Treatment of Laryngeal Cancer. The Boston Medical and Surgical Journal, published July 26, 1917.

The Technic and After-Treatment of Hemilaryngectomy and Total Laryngectomy. The Journal of the American Medical Association, Vol. LXIX, pp. 863-868; published September 15, 1917.

Surgical Treatment of Laryngeal Cancer With an Analysis of Seventy Cases. New York State Journal of Medicine, published October, 1922.

Operation of Total Laryngectomy for the Cure of Intrinsic Cancer of the Larynx. ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, published December, 1922.

The Operative Treatment of Cancer of the Larynx. The Journal of Laryngology and Otology, published February, 1924.

Cancer of the Larynx. Published July, 1926.

Nasopharyngeal Atresia. Archives of Otolaryngology, published July, 1927, Vol. 6, pp. 1-27.

An Operation for the Relief of Abductor Paralysis of the Larynx. Archives of Otolaryngology, published July, 1928, Vol. 8, pp. 37-46.

Laryngeal Cancer—Early Diagnosis and Treatment. Archives of Otolaryngology, published March, 1929, Vol. 9, pp. 237-244.

Some of the Complications of Laryngectomy. The Laryngoscope, published October, 1929.

The Operative Treatment of Cleft Palate—A New Method. Archives of Otolaryngology, published November, 1929, Vol. 10, pp. 491-512.

Cancer of the Larynx—Report of Several Unusual Cases. Archives of Otolaryngology, published December, 1929, Vol. 10, pp. 585-602.

Analysis of Cases of Laryngeal Carcinoma Seen Since October, 1929. Laryngoscope, published June 16, 1930.

Upper Respiratory Focal Infections in Some of Their Regional and General Manifestations. Bulletin of the New York Academy of Medicine, published February, 1931, second series, Vol. VII, No. 2, pp. 71-109.

Infections of the Upper Respiratory Tract in the Etiology of Uveitis. Read before the Association for Research in Ophthalmology at Philadelphia, June, 1931. To be published in the Transactions of the Association for Research in Ophthalmology for 1931.

NOTICES.

The next annual meeting of the American Laryngological, Rhinological and Otological Society will be held in Atlantic City, N. J., May 23, 24 and 25, 1932.

The meeting of the American Academy of Ophthalmology and Otolaryngology will be held in Montreal, September 19th to 23rd. Headquarters will be the Mount Royal Hotel.

The American Board of Otolaryngology will hold an examination Saturday, September 17th, at the Royal Victoria Hospital. Prospective applicants should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for proper application blanks.

The Indiana University School of Medicine offers a two weeks' intensive post-graduate course in otolaryngology from April 18th to 30th, inclusive.

This course will be conducted, and all didactic, and anatomical work will be given, and personally supervised by Dr. John F. Barnhill. The course will be supplemented by clinical demonstrations, and operations during the morning hours by members of the Otolaryngology staff, and Doctor Barnhill, at the University Hospitals.

The hours from 8:00 until 11:00 a. m. will be occupied by clinical work in the University Hospitals. The hours from 1:00 to 5:00 and 7:00 to 10:00 p. m. will be spent in the anatomical laboratories. This makes a total of 84 hours of anatomical study and 36 hours of clinical instruction and observation.

The fee for the course will be \$75.00, which covers the cost of anatomical material. For full information concerning the course, address all communications to Dr. W. D. Gatch, Dean, Indiana University School of Medicine, Indianapolis.

Books Received.

Food Allergy, Its Manifestations, Diagnosis and Treatment with a General Discussion of Bronchial Asthma.

By Albert H. Rowe, M. S., M. D., Lecturer in Medicine in the University of California Medical School, San Francisco, California; Chief of the Clinic for Allergic Diseases of the Alameda County Health Center, Oakland, California; Consultant in Allergic and Metabolic Diseases, Highland Hospital; President of the Association for the Study of Allergy, 1927-1928. Cloth. Price, \$5.00. Pp. 442. Lea & Febiger, Washington Square, Philadelphia, 1931.

This monograph on Food Allergy represents the results of careful, ingenious and original observation. It represents one of the most important advances in our knowledge of the diagnosis and treatment of allergic diseases. In the past, the successful management of the allergic patient has been disappointing because too much reliance has been placed upon the results of skin tests and too little attention has been directed to food as a cause of allergic reactions. In a very comprehensive manner the author outlines the principles of treatment and diagnosis based upon the use of "elimination diets." The intention of these diets is to eliminate the most common food factors, which are wheat, eggs and milk. Other commonly eaten foods are also eliminated. In this manner the possibility of eliminating the offending allergens from the diet is highly probable. When the patient becomes symptom-free, foods are gradually added to the diet until symptoms are produced. The offending foods are thereby discovered. The volume is replete with references to all the important work done on food allergy as well as to other phases of the subject. Many interesting case reports throughout the book illustrate the practical application of the elimination diets. After reading this most interesting contribution one is greatly impressed with the important relationship of food allergy, not only to otolaryngology, but to all branches of medicine. Anyone interested in the management of the allergic patient will greatly benefit by the important knowledge that has been contributed in this monograph.

F. K. H.

Asthma and Hay Fever. In Theory and Practice.

Part I: Hypersensitiveness, Anaphylaxis, Allergy, by Arthur F. Coca, M. D. Part II: Asthma, by Matthew Walzer, M. D. Part III: Hay Fever, by August A. Thommen, M. D. C. S. Thomas, 220 East Monroe Street, Springfield, Illinois, 1931.

In this book Coca discusses hypersensitiveness, anaphylaxis and allergy; Walzer presents the subject of bronchial asthma, and Thommen that of hay fever.

In Part I, Coca presents the immunological aspects of hypersensitiveness, anaphylaxis and allergy and reiterates the distinction between allergy and atopy. A very useful chapter on the preparation of extracts and solutions for use in testing and treatment is included in this part.

Part II is a most comprehensive discussion of bronchial asthma. All the controversial phases of the asthma problem, clinical as well as immunological, are fully discussed. The chapter on atopens and other excitants is of inestimable value, because it assists the clinician in the practical application of the information gained by cutaneous testing. It contains all the pertinent information concerning the common atopens and excitants.

Part III presents the history, the etiology, the mechanism, the symptomatology, the diagnosis and the nonspecific and specific treatment of hay fever. Considerable space is devoted to its botanical aspects, which is aided by many illustrations.

The bibliography contains over 2,000 references, attesting the thoroughness with which these three subjects are discussed. An excellent index, as well as special index for atopens and excitants, makes all this data easily available.

C. H. E.

Recent Advances in Allergy: (Asthma, Hay Fever, Eczema, Migraine, etc.)

By George W. Bray, M. B., Ch. M. (Sydney). With Foreword by Arthur F. Hurst, M. A., M. D. (Oxon.), F. R. C. P. 98 Illustrations including 4 Colored Plates. P. Blakiston's Son & Co., Inc., 1012 Walnut Street, Philadelphia, 1931.

This book is a compendium of the trend of research and the present knowledge in allergy and allergic manifestations. The data is obtained from a very extensive bibliography and is given with the critique of the author in a concise and clear manner.

In discussing allergy, separate chapters are devoted to the biochemical aspects; changes in the blood; climate, altitude and

environment; the toxic factor; the nasal factor; the endocrine factor; the nervous or psychic and hereditary factors; protein skin reactions; patient's history, examination and experimental observations. One infers that the author favors a biochemical explanation for the fundamental basis of allergy.

In the clinical section of the book, asthma; pollen, cutaneous, food, drug, bacterial and physical allergy; cerebral manifestations; serum reactions and other manifestations such as enuresis, eye and joint reactions; sensitivity to molds, fungi, insects and parasites, are discussed in separate chapters.

The increasing number of papers on this subject, appearing in all the medical journals of the world, makes it difficult to keep fully informed. This accurate and complete résumé presents this mass of information in compact form. In addition, it gives the clinical experience of the author with asthma and allied conditions in children.

C. H. E.

Abstracts of Current Articles.

Avertine Narcosis per Rectum (Étude de la narcose rectale a l'avertine).
Prof. P. Guns (Louvain). Ann. d'Oto-Lar., 1:981, Sept., 1931.

Reporting twenty-five cases, using 3 per cent avertine solution at the average rate of 10 centigrams per kilogram of body weight, secured good narcosis in only 44 per cent. In spite of preliminary use of morphin, it was necessary to add novocain or chloroform, according to the case (mastoids, external sinus work). Marked reduction of blood pressure, approaching heart collapse in some cases, was frequent and could not be rapidly lifted, even by intravenous caffen. Camphorated oil was useless. And if the blood pressure does not go down, the patient is sure to need other anesthetic agents. Although patients go to sleep on the operating table four to five minutes after the narcotic enema, they are likely to awaken at the first incision. F.

Acute Otitis Media in Infants and Young Children during 1930-1931.
A. J. Cone (St. Louis). Arch. Otolaryng., 14:797-814, December, 1931.

Cone, in the St. Louis Children's Hospital, treated 194 cases of otitis media out of 402 cases admitted to the infants' ward with a diagnosis of a general systemic condition including pneumonia, septicemia, meningitis, pyuria, nephritis, secondary anemia, diarrhea, birth injuries, congenital defects, asthma and others. Spontaneous discharge occurred in 43. Four hundred myringotomies were done in 174 cases. The mortality in the operative cases was 35 per cent, and all cases combined, 32 per cent. Only occasionally did ear cultures correspond to mastoid cultures. The predominating organism from the ear cultures was staphylococcus aureus, while the mastoid usually showed hemolytic streptococcus. Forty-seven of the 194 cases of otitis media had diarrhea. The efficacy of mastoidectomy or nonoperative results is somewhat undecided because of various associated systemic conditions. but myringotomies seem to be of a great help in nonoperative cases, whether the ear was primary or secondary. The diarrhea and vomiting he believes to be due to a metabolic change, and changes in the gastric acidity of such nature as to allow bacillus coli to live in the stomach and upper intestinal tract. G. T.

Effects of Some Drugs on the Vestibular Response to Rotation.

Ross, et al. Arch. Otolaryng., 14:755-775, December, 1931.

Atropin, nitrite, morphin, pilocarpin, picrotoxin, caffen, bromide, camphor, nicotin, cocain, apomorphin and strychnin were used on dogs. Duration of nystagmus after rotation was decreased somewhat by sodium bromide, picrotoxin, apomorphin and caffen but to a greater extent by strychnin. This effect of strychnin plus the fact that high spinal anesthesia and severing of the columns of Gall and Burdoch led to increased labyrinthine irritability made these authors conclude that the spinal cord exerts an inhibitory action on the vestibular apparatus.

G. T.

Tumors of the Tonsil and Pharynx (357 Cases).

New and Childrey, Mayo Foundation. Arch. Otolaryng., 14:699-713, December, 1931.

The authors present a very thorough review of a study of 357 cases of tumors in tonsils and pharynx. As a result of this study they conclude: The squamous cell epitheliomas in this situation are almost five times more common than all other malignant tumors. Sarcomas in the pharynx are principally of the lymphosarcoma type. Next in order of frequency is the fibrosarcoma commonly occurring in the nasopharynx. Adenocarcinomas of the mixed tumor type are found in 74 cases. They also enumerate the hemangio-endothelioma, melano-epithelioma, myeloma and malignant dermoid. Metastasis to the cervical lymph nodes occurs early in epithelioma and lymphosarcoma. Epithelioma may be confused with Hodgkin's disease, particularly if found in the nodes bilaterally, as often occurs. Cervical metastasis is so common that in every case of enlargement of the neck, that cavity of the pharynx should be searched carefully. The average duration of all the tumors studied in these cases was seven and four-hundredths months. The miscellaneous group included two cases of long duration, a melano-epithelioma had apparently been present five years and a hemangio-endothelioma twelve years. Symptoms and signs of malignant tumors of the pharynx, as a rule, attract little attention until the tumor has reached a considerable size. Seventy-five per cent of the cases' early symptoms were referred to the throat. These consisted of paresthesia, local irritation, sensation of fullness or the presence of a foreign body,

as well as soreness or pain in the throat. Few cases cause hoarseness or thick speech. Salivation and dysphagia were common. They feel that biopsy is one of the most valuable aids in diagnosis, but should be followed shortly by treatment. The average age of all patients was 54.7 years. Except for lymphosarcoma, malignancy is rarely primary in the tonsil itself. As to treatment they conclude that lymphosarcoma is more radio-sensitive than epithelioma; that certain epithelial growths of highly cellular, undifferentiated types are markedly radio-susceptible; and that irradiation is unquestionably the treatment indicated for sarcoma of the pharynx or tonsil. Depending upon the situation and extent of the lesion, epitheliomas of the tonsil or pharynx are treated with combination of irradiation and surgical measures, cautery or diathermy, namely, squamous cell epitheliomas graded 3 and 4. Irradiation also has been used both outside the neck and directly into the wound in the pharynx. They conclude the prognosis following the treatment of epitheliomas and sarcoma of the tonsil and pharynx is questionable. The length of life is placed at from one to two years by most observers. But the authors show a surprisingly large number of living patients of three, five and seven year cures.

G. T.

Postoperative Complications of the Radical External Operation on the Frontal Sinus.

Harold I. Lillie (Rochester, Minn.) *Arch. Otolaryng.*, 14:793-797, December, 1931.

In this article a choice of anesthesia is discussed as well as indications for the Killian or Lynch operation. In a series of 158 consecutive cases postoperative complications developed in 45. The most common was diplopia, but frontal lobe abscess, meningitis and orbital abscess did occur.

G. T.

The Relation of the Nasal Sinuses to Asthma.

H. G. Tobey, (Boston). *Arch. Otolaryng.*, 14:784-792, December, 1931.

The author reviews the work of James Adam, Haseltine, Osler, Rackemann and others. He believes that toxemia may be the underlying cause of asthma while admitting that the definite cause is unknown. Reflex asthma he recognizes, but no definite type of nasal disease does he find to act as a "trigger mechanism" to set off an attack of asthma.

G. T.

Percaine in Oto-rhino-laryngology (La percaïne en oto-rhino-laryngologie).

Dr. M. Prévot (Marseilles). Rev. de L. O. R., 52:209, March, 1931.

Percaine (chlorhydrate of the diethylethylenediamide of butyl-oxyinchoninic acid) is unrelated to cocain, novocain or storain. Prévot recommends the following formula for paracentesis:

RX	Percain	4	5
	Chloral		
	or		
	(Chloretone)	25	5
	Menthol	30	
	Phenol	30	

For surface anesthesia of the nose, 2 per cent solutions; for the throat and larynx $1\frac{1}{2}$ to 1 per cent solutions are recommended. None of the ischemic action of cocain is present; adrenalin is required, as with butyn, to keep down a slight tendency to local congestion. One and one-half per cent solution facilitates laryngeal biopsies, removal of papillomata, etc.

For injection 1:1000 and 1:500 are recommended, permitting painless skin suturing after facial work, lasting much longer than the effects of most injected agents. Postoperative pain after tonsillectomy is long deferred and reduced. The author suggests the same caution as in the use of butyn and novocain, but has thus far had no bad results from toxic absorption.

F.

Society Proceedings.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting Held on Monday, October 5, 1931.

THE PRESIDENT, DR. HOWARD C. BALLENGER, IN THE CHAIR.

The Mechanics of Audition.

DR. A. G. POHLMAN.

(Author's Abstract.)

Audition is dependent on the conduction of vibrations which lie within the audible range to the end organ, and on the transformation of the stimuli at the end organ into action currents which are translated by the brain into terms of the sources. It is well known that the development of the vertebrate ear seemingly violates the physical requirements essential to an organ of great sensitivity because the perceptive apparatus is an immersion end organ and because air sounds are almost completely reflected back in their conduction to water. The development of an immersion end organ, however, fulfills the physiologic requirement of placing the perceptive apparatus under conditions of acoustic insulation which permits the possibility of great sensitivity in response. The acoustic insulation is overcome through the development of a middle ear, which acts as a transformer to match the resistance differences in the two media. The evidence seems to show that the middle ear apparatus is functionally more efficient in the conduction of sounds of weak intensity and that as the loudness of the signal rises, the intensity of reaction in the internal ear becomes proportionately less. This type of transformer is accordingly one of limited intensity reaction and affords protection to the auditory end organ in acting as a shock absorber on intense sounds. The experimental evidence also indicates that the middle ear apparatus is about equally efficient in the conduction of air sounds of all frequencies to the internal ear, as opposed to the generally accepted idea that the conduction through the ossicles is limited to low frequencies. The problem of middle ear me-

chanics has been obscured through the attempts to interpret unreal phenomena in audition, such as paracousis Willisii and prolonged bone conduction. The prolonged bone conduction is a real phenomenon only under conditions where the perceptive apparatus is normal, the sound conduction system functional and the drum membrane loaded or the external canal occluded. Under other conditions bone sounds are conducted directly through the wall of the otic capsule and irrespective of the mechanical conditions of the middle ear. This interpretation establishes the value of the comparison between air and bone acuity in differential diagnosis.

The speaker opposed the usual interpretation of conduction deafness and holds that the accepted explanation is dependent on the degree of presbycusis in the examiner; the inadequacy of the test methods before the invention of the audiometer, and on the sensitivity of the internal ear as a function of the frequency. The experimental evidence also opposes the accepted idea that the ossicular chain acts as a lever which decreases the amplitude and increases the force with which the drum membrane vibrations are propagated to the labyrinth. The comparative anatomy suggests that a leverage action is present in all forms with middle ear apparatus and that this leverage acts as a shock absorber against a displacement due to topographic changes in the region. There is no evidence that the intrinsic muscles act as an accommodative system, and while the *M. stapedius* in man has been observed reacting to sound stimulation, this is not the case in the *M. tensor tympani*. The middle ear apparatus may accordingly be interpreted as one with efficient conduction of audiovibrations of all frequencies and most efficient at low intensities. It may also be regarded as a shock absorbing mechanism.

The morphology of the internal ear has been generally interpreted in terms of the mechanical analysis of pitch and predicated on the assumptions originally made by E. H. Weber that the otic capsule is inelastic, the liquid is incompressible, and the round window is the only compensation opening which permits displacement of the stapes. It is upon these three premises that all theories of pitch analysis which call for transverse vibrational responses in the cochlea duct and its contents have been based.

The three premises, however, are not facts, and the evidence seems to substantiate the idea that the morphology of the internal ear has been misconceived. The evidence indicates that the round window is not related to the function of sound analysis.

The explanation as proposed seeks to eliminate the indirect activation theories and substitute one of direct activation where the auditory cells themselves respond directly to the vibrations passing on or through them. This explanation suggests that the serial arrangement in the end organ is dependent on the development which also accounts for the dimensional differences in the cochlear duct and its contents. The experimental evidence favors the idea that morphology may be interpreted as a protective mechanism against transverse vibrational displacement in the internal ear.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH: I am much pleased with Dr. Pohlman's able presentation of the problems of hearing.

The problems of hearing are rather intriguing, chiefly so because the reactions are physical and seem, therefore, more capable of analysis than are those of light perception, for example, for we grasp the physics of hearing more readily than we do the chemistry of seeing.

One hardly knows just how to begin a discussion of Pohlman's paper; also one probably will have difficulty in knowing just where to stop. According to Pohlman's criticisms we have very little basis for any theory of hearing, but, as a matter of fact, we do hear in spite of these criticisms and hear with the organ of hearing.

Two factors are necessary in the physiology of hearing. The first is the existence of a mechanism for collecting sound waves which originate in the outer air and conducting them into the head where they come in contact with the endings of the eighth nerve. The second is a mechanism for transforming these physical sound impulses into nerve impulses, and the transmission of these nerve impulses to the centers in the cortex where they are interpreted as sound.

We are quite satisfied about the function of part of this so-called sound conducting mechanism. The auricle, for example,

is the mechanism for collecting the sound waves and directing them into the external meatus, but it is of relatively little use in man. It seems to be much more important in many lower animals. Again, the external meatus is the means for getting the sound waves into the head. It is not such an important mechanism, for if you put your fingers in your ears and block off the external canal, everyone in this room will hear what I am saying without raising my voice. In the same way, a child born with atresia of both external auditory canals experiences very little difficulty in hearing, provided the developmental anomaly does not also involve the inner ear. The ordinary voice is heard easily the length of this room by such a child.

It should be clear that operations to open up the external auditory canal in order to allow sound waves to reach the labyrinth are not necessary, because if the labyrinth is involved such an operation would not be of use, and if the labyrinth is not involved the operation is not necessary.

The next step is a more difficult one: the transference of the air-borne sound impulses into the fluids of the labyrinth. It is generally believed that the drum membrane and the chain of ossicles serve as a series of levers for increasing the intensity of the sound impulses which impinge upon the drum membrane and finally reach the fluids of the labyrinth by way of the stapes footplate. Some idea of the increase in intensity that is brought about by this chain of ossicles can be visualized by comparing the size of the drum membrane with that of the footplate of the stapes.

The clinician becomes rather skeptical about the value of this leverage mechanism. Anyone who has had much of a background of clinical experience has encountered this situation: drum membrane gone, chain of ossicles exfoliated, stapes intact, where the defect in hearing is so slight that it is scarcely appreciated by the patient. A careful test will find some hearing defect, but this may be very slight, so that this mechanism for transferring the air-borne impulses to the fluids of the labyrinth cannot be of very great importance.

The important problem is the question of how these physical sound impulses get into the labyrinth. Is it through the chain of

ossicles by way of the footplate of the stapes in the oval window or is there another route by way of the round window and *membrana tympani secundaria*? The practicing otologist has before him a mass of evidence on the interference of sound waves reaching the labyrinth when there is anything impairing the mobility of the stapes. Fixation of the stapes does not interfere with the transferring of the sound waves to the labyrinth and interferes more with the transference of low pitched notes than with high pitched notes. The high pitched notes are somewhat interfered with, but by no means to the same degree as are the low pitched notes. This is an everyday experience which the practicing otologist encounters.

The question comes up here whether the high pitched notes may not be conducted into the labyrinth by some other route than by the chain of ossicles, and the suggestion has been made that the low pitched notes reach the labyrinth by means of the conducting mechanism and the oval window, whereas the high notes reach the labyrinth by the route of the round window. But there is an objection to this hypothesis: There must be some place in the tone scale where low pitched notes and high pitched notes are conducted equally well by the oval window and by the round window. This would create an impasse which, as a matter of fact, we do not experience in our hearing for any part of the tone range.

There is a very simple explanation of why the low pitched notes have more difficulty in being transmitted by stapes the movements of which have been impaired than have the high pitched notes, and it is simply that the vibrations of the stapes produced by the low pitched note are of greater excursion than are those of the high pitched note. The higher one goes in the tone scale, the less these excursions become, and even complete bony fixation of the stapes does not seem to interfere with the passage of the high pitched note directly through solid bone to the fluids in the labyrinth.

There was an interesting experiment carried on by Siebenmann which threw a sidelight on this problem of sound conduction. We are all familiar with the results produced by overstimulation

from high pitched whistles in a guinea pig confined in a box. These degenerations of Corti's organ were not produced when the ossicular chain was broken by the resection of the long process of the incus, for example.

The mechanism for transforming the physical impulses of sound to nerve impulses is, as I have said, a necessary part of the function of hearing. There are two approaches to the solution of this problem. One is to study the phenomena of hearing as, for example, tone analysis, and then try to visualize some sort of mechanism in the internal ear which can function so as to account for peripheral tone analysis. The physical resonators constitute the mechanism which seems most suitable for performing this function. With a physical resonator in mind, one examines the cochlea to see what structure is there that can perform such a rôle. This has been the method that is usually adopted. It is the plan followed by Helmholtz. He was impressed by the phenomenon of physical resonators as a means for tone analysis, and inquired of anatomists for a structure in Corti's organ which could fill this rôle. He selected the rods of Corti as the rod resonator, these varying in size from one end of the cochlea to the other, he felt might act in this manner, responding in different areas to tones of different pitch, and by jarring the neighboring hair cells bring about their stimulation. Just how this stimulation of the hair cells by jarring was accomplished was not elucidated. He gave up this idea of the rod resonators because birds and alligators, for example, did not have rods but, nevertheless, could hear very well, and he selected the basilar membrane, the radiating fibers of which vary in length from one end of the cochlea to the other, as a physical resonator, because he thought it might possess the physical properties of a string resonator. As a matter of fact, a careful study of this membrane reveals very definite objections to its acting in this manner. For example, as we approach the lower end of the basal coil the membrane loses entirely its resemblance to a string resonator and becomes a thick, wedge-shaped structure and quite incapable of filling the rôle assigned to it. The fact that it is a vascular structure shows that it is not capable of vibrating at all times in the same manner to the same impulses.

There is a more fundamental way of approaching the problem of deciding how the transference of physical to nerve impulses takes place, and that is to study the structure of the organ itself, and see whether we can reach a conclusion as to what the particular function of the various parts of Corti's organ may be and then to build up a theory which will best explain the phenomena of hearing. This reasoning is very simple. The hair cells are undoubtedly the mechanism in which the transference from physical to nerve impulse takes place. The stimulation of the hair cells is unquestionably accomplished by irritation applied to the projecting cells. This irritation cannot reach the hairs by impulses passing through the endolymph because the hairs do not stick up freely into this fluid but are attached to the under surface of the membrana tectoria. I was the first to demonstrate this important anatomic relationship in 1907. Others in this country soon verified this finding, and more recently Held and Wittmaack have both verified the same condition.

It is clear, therefore, that stimulation of the hair cells must be accomplished by some interaction between the membrana tectoria and the projecting hairs. There are two ways by which this interaction may be brought about. One is by movements of the membrana tectoria itself and the other by movements of the membrana basilaria. From a biologic standpoint, it is the membrana tectoria which is the logical structure for responding to impulses in the endolymph. In the first place, this brings the reactions of the cochlea in accord with the reactions in the two other types of end organs in the labyrinth, the macula and crista acustica, in which the superimposed structures, the otolith membrane and the cupola, are the structures which move to stimulate the hair cells. In the second place, sound impulses reaching the labyrinth via the oval window impinge first and most forcibly upon the membrana tectoria. The membrana vestibularis (Reissneri) is such a delicate, film-like structure that it offers no interference to impulses passing directly through the scala vestibuli to the scala media.

The question arises, what is the manner of response in the membrana tectoria? This structure varies in size from one end of the cochlea to the other, and its size must influence the physical

response to impulses passing through the endolymph. To say that it can or cannot react in a particular way is unwarranted, since we know so little about the structure of the membrana tectoria. It is an elusive membrane. My feeling is that we should accept that kind of response in the membrane which will best explain the phenomena of hearing and that the efforts to fix upon the membrana basilaris a function which biologically it is not intended to perform is misdirected.

Models have been constructed to show how the structures may respond to sound impulses. For example, Hardesty builds up a model which he thinks resembles the membrana tectoria and actually demonstrates that it responds in different parts to tones of different pitch. Recently an Englishman has developed a model which he assumes demonstrates what the responses of the membrana basilaris would be provided it responded according to the principle of physical resonance. Ewald developed a model to show how undulations throughout the membrana basilaris represented the type of responses of this membrane to sound impulses.

My feeling is that anyone who has studied the actual structure of the organ of Corti through the microscope would hardly feel like attempting to create any model which could be taken seriously as demonstrating how structures in the cochlea perform their function. Hardesty's model, for example, no more resembles the membrana tectoria than does a tapering haystack; Ewald's model bears no more resemblance to the membrana basilaris than does a stretched sheet; and the model which I referred to above demonstrating the response of the membrana basilaris according to the principle of physical resonance demonstrates only what we all knew before, that such a model should respond to the principle of physical resonance, but his model is no demonstration of what the membrana basilaris might do.

Pohlman points out objections to Ewald's theory but fails to point out the most fundamental of all objections, namely, that the attachment of the hair cells to the under surface of the membrana tectoria makes it impossible for certain groups of hair cells occupying the crests of supposed wavelike undulations in the membrana basilar to be stimulated separately. The anatomic rela-

tions between the hair cells and tectorial membrane make it necessary that every undulation of the membrana basilaris stimulates, not only the hair cells which occupy the crests of these undulations but all the hair cells throughout Corti's organ, and would thus, as Ewald himself points out, render peripheral tone analysis impossible.

I think these discussions of the principles underlying physiology of hearing are interesting and are more or less profitable, but not in the sense that they solve for us in any satisfactory manner any particular clinical problems. I quite agree with Pohlman that operations aiming to open a passage for sound impulses to enter the labyrinth by boring through the labyrinthine wall can hardly be expected to give any satisfaction. In the first place, to carry out an operation for making a fistula of the bony capsule of the labyrinth without breaking through into the cavity allowing the escape of perilymph and endolymph, is, to say the least, a very precarious undertaking, for if the fluids of the labyrinth escape the function is entirely destroyed. In the second place, it must be an exceptional situation where an opening made in the bone will not close spontaneously and thus destroy any possible good effects from the operation. And finally, the operation would only be undertaken in cases of profound deafness, and in these cases the profound deafness is not so much the result of stapes fixation itself but of secondary degeneration of the nerve, which cannot be benefited by making a fistula into the labyrinth. My feeling is that if the facts were placed squarely before a patient on whom we proposed to undertake an operation of this kind, he would decide not to have it done.

DR. ALFRED LEWY: I once listened to Einstein, expounding his theory, and I thought I caught a glimmer of it. Later I heard the theory explained by Professor Lunn at the University of Chicago, and I thought I had gotten a little more out of it. But when I tried to tell anybody else what I thought I understood it was impossible to do so.

I feel very much the same in connection with Dr. Pohlman. I heard most of what he said this afternoon, and this evening I was so entranced, in listening to what he had to tell us, that I forgot to put down any notes until after he got through speaking.

However, he apparently demolished most of our ideas so far as the theories of the perception of sound are concerned. For some reason, he didn't touch upon the old and perhaps now discarded telephone theory. I would like to have him, if he can, tell us what is the matter with that. We have been told a number of times what is the matter with it, but I would like to have his version.

Rather than discuss his paper, I shall ask him a few questions. As I indicated, I do not feel competent to discuss it. How does he know what the range of hearing in the parrot is? I know he judges it by what he calls the motor output, by which I think he means the range of sound that a parrot is capable of making. But it has been found possible to obtain in animals certain reflexes of the eye or some other action that would indicate whether they do or do not hear a certain sound. I would like to ask Dr. Pohlman whether that has been carried out in connection with the parrot or with any other animal in which the structure of the cochlea is different from that of the human being.

Not being a physicist, I don't know how they go about finding it out. I would like to ask him whether the mechanical vibrations that have been transmitted through the ear mechanism may have an electrochemical effect upon the nerve endings. Exposition of a philogenetic history of the cochlea raises the question of whether some of the neuro-epithelium, the original neuro-epithelium, may be attuned to an electrochemical effect that is connotated by that particular pitch. The latter developments may be attuned to a different pitch.

Dr. Pohlman promised us this afternoon that he would tell us what his theory is. I didn't understand that he expounded any theory of his own this evening. There is one thing that has always troubled me. I am troubled in understanding exactly why sound waves are called waves. If they are areas of expansion and rarefaction, contraction in the air, why, in the teaching of sound waves, didn't they draw a picture of an interrupted series of dashes of equal length to represent frequencies and of different dashes to be of different degrees of thickness to represent different degrees of energy; in other words, greater amplitude. I

don't know, but it seems to me if we had it explained in that manner it would be a little more easily grasped.

DR. ROBERT SONNENSCHN: It is very difficult to try to measure up in any way in discussion with a man like Dr. Pohlman, who is a good physicist, a good acoustician, and a very clever talker. We have listened to a great many fundamental, important facts regarding physics and, to a certain extent, the physiology of hearing, this afternoon. The various theories of hearing I know very little about. I know the names of some of them: Helmholtz's peripheral analysis theory, the telephone theory, central analysis and volley theory, and so forth.

It seems to me the highly scientific aspects of the subject which have been stressed so much perhaps warrant calling attention to a few of the things Dr. Pohlman mentioned this afternoon, and to a slight extent this evening. He calls attention to the fact that there are certain unreal phenomena, such as paracousis Willisii. My attention was called by the essayist to the first edition of Troeltsch's book, which appeared in 1862, in which he states that the individual who is hard of hearing is not masked by the loud sounds about him at the same pitch at which he is deafened, and, therefore, the speaker, raising his voice, because he is masked by these sounds, speaks much louder, and the individual with the conduction impairment hears. Dr. Shambaugh did some excellent work on paracousis with the audiometer. I think it is important to stress that, because you still find very excellent otologists with divergent opinions. No less a man than Professor Alexander, the last time I was in Europe, said he could prove, on the street car and other places, that it was an actual phenomenon.

The point has been brought out that the individual who apparently has an increased bone conduction, at least what we call prolonged bone conduction, when tested in an office, does not have an increased bone conduction when tested in the silent chamber. Dr. Pohlman has shown that there is such a thing as actual increase in bone conduction, when you load the drum membrane or occlude the external auditory canal.

I have always maintained it is not the best policy to test the patient with your own bone conduction. That presupposes that

your own bone conduction is normal. It is better to have an objective standard which you intend to use in your tests, having determined by examination of, let us say, 100 apparently normal individuals. The bone conduction varies, let us say, fifty seconds when used in a certain manner; then you can know, from practical experience, that when one individual hears only thirty seconds his bone conduction is diminished. If the next one hears 100 seconds, it is increased.

It does not make much difference whether that bone conduction is increased from the scientific standpoint, because we, as a rule, must examine patients in noisy places. Therefore, if we do the tests in a certain manner and know the standpoint, we have the criteria from a practical standpoint.

With reference to the absolute bone conduction test, about which we read a good deal in the English literature, placing the finger in the ear and producing the prolonged bone conduction by occluding the auditory canal. This is the same principle as one type of the old Bing test. In normal individuals bone conduction is increased, but in an individual with Rinne negative reaction and lengthened bone conduction, occluding the ear no longer increases the bone conduction. I mention it because there is so much confusion on that subject.

Dr. Pohlman, if I quote him correctly, stated in an article last year that the air sound transmission apparatus appears to be of equal efficiency throughout the audible scale. If an individual hears any frequency at normal intensity, he may be regarded as not having conduction deafness. Of course, that is true, the same as the statement he made this afternoon. When you test an individual with the audiometer, you find he has diminished hearing for the low tones; he also has diminished hearing for the high tones. If he has a conduction impairment, he also does not hear the high tones well. That, no doubt, is true, when it comes to finer quantitative measurements. But if in actual practice that fact does not hold true, you are not testing accurately, but testing in a practical manner, by means of the voice. And, as we have stated so often, even though the voice cannot be standardized as regards intensity or pitch, especially intensity, and the fact that the dif-

ferent voices vary qualitatively because the overtones in the speech give the peculiar quality to the different individuals, nevertheless, if we use the test in the proper manner, we get valuable findings. For instance, if you take a patient and stand him off twenty feet and whisper low tones, you find one individual does not hear the low tones until you get within a meter. But when you use the high pitched tones he often hears at five or six meters. We know that is a beginning conduction impairment. The high tones tested with the audiometer may show some contraction. But those are the very tones about which he spoke this afternoon, that we are not conscious of hearing anyway.

I am as much interested as anyone in knowing the scientific facts regarding anything, but we should bear in mind the fact that various theories of hearing are not only not settled, but in greater confusion than ever before. Every year we are getting modifications of hearing theories, just as in therapeutics, where for a certain disease ten, fifteen or twenty remedies are suggested. But you may be sure none is very efficient, because if one were, we would stick to that one. In syphilis, we rely on mercury, arsenic and the iodids. We do not use twenty or thirty different things.

DR. J. HOLINGER: It seems to me that Dr. Pohlman is going too far in rejecting every attempt at explanation of the act of hearing. This nihilism is unfortunate, because it creates in many heads a state of mind which says, "What is the use of bothering?" and it paralyzes interest and real study. This is so much more justifiable at the present time when the study of many observations on the battlefields of the great war shows so many coincidences with the results of experiments made on animals fifteen and twenty-five years earlier. I cannot understand how anybody can doubt the interaction between the tectorial membrane and the hair cells when it is shown in experiments of over irritation of birds' ears that the tectorial membrane can crush and smash hair cells and only those which it can reach while those beyond its reach are intact. All these points have been discussed before this society so many times that I flatter myself most of our Chicago colleagues know the point beyond which they cannot follow Dr. Pohlman. For example, the tectorial membrane is a tissue

floating in the mobile fluid of the labyrinth and a little more viscous than the fluid itself and therefore very apt to react to every movement of and in the fluid. If we try to understand the experiments on birds from this viewpoint, the effect of the round window appears in another light. The interior of the labyrinth is elastic and not inelastic, as Dr Pohlman says. This changes our standpoint as to the function and action of the round window. Deafness as well as intact hearing has been found in ears where the round window was more or less immovable. Our clinical experiences are certainly not final, and to go further than this is putting too much weight on uncertainties.

DR. A. G. POHLMAN (closing): The function of the auricle in the human being appears to be restricted largely to reflection of the higher frequencies, and its efficiency seems to be greatest at or above 8000 Hertz. Increasing the size of the auricle through placing the hand back of the ear lowers the frequencies which it will reflect efficiently. One sees the effect of the auricle in tests at high frequencies with the audiometer where the patients will tell you they hear a given tone better when the receiver is removed a short distance from the ear.

I neglected to mention a point which may be of interest to the otologists. When we test a case with the audiometer and establish a curve, we may conclude that this individual has lost a certain per cent of his useful hearing. Useful hearing, however, to the patient usually means useful hearing in so far as conversational purposes are concerned. While a great deal of work has been done with the masking effect of one pure tone on another pure tone, the experimental aspects on a masking of low frequency components on the high frequencies in a complicated sound, as in speech and the effect on the intelligibility, has not been accurately determined. The audiometric curves are therefore not necessarily a criterion of the functional efficiency so far as the spoken voice is concerned without more definite evaluation.

Degeneration experiments on the cochlear end organ have been repeatedly accomplished. It is, however, perfectly reasonable to suppose that the auditory cells undergo fatigue just as any other highly organized cell. To say that a given area of the cochlear end organ has degenerated on account of a prolonged exhibition

of a loud sound does not mean that either the basilar membrane or the tectorial membrane has been mechanically responsible for the lesion. The cell area might have degenerated through excessive stimulation irrespective of how this stimulation was brought about. The evidence is therefore not conclusive that the activation of the auditory cells is extrinsic.

Dr. Shambaugh referred to the development of the otolithic membrane and otoliths. It must not be forgotten that the functional relation of the otoliths to the otolithic membrane is one of displacement to stimuli of slow motions, and I fail to see how the tectorial membrane without otoliths might react to high frequency vibrations of extremely small amplitudes.

Dr. Lewy asked about the telephone theory, which, in my opinion, is not a theory at all, because it explains nothing and assumes a central analysis which is seemingly contrary to the facts. If one carbon button or a single condenser transmitter can handle practically all audiofrequencies through a wide intensity range, it is difficult to see why Nature should have supplied the internal ear with say 20,000 auditory cells unless a peripheral analysis is indicated. Rutherford, like other physicists, may have proposed his conception with no very definite ideas on either the morphology or the physiology of the apparatus.

As to the range of hearing in birds, it may be that the Weaver and Bray technic will establish this quite definitely. I believe one must admit that the mechanics of hearing in birds must be similar to that in mammals. A mocking bird, which imitates the creaking of a rusty hinge, must hear at least to the neighborhood of 10,000 vibrations if it is reproducing what it hears; otherwise the creaking noise would not sound quite normal to a human being. It has been demonstrated that the noise produced by the jingling of a bunch of keys changes in quality if vibrations above 8000 are filtered out. If the mocking bird can imitate the creaking of a gate hinge, I for one am prepared to assume that it hears at least the frequency range of the human being.

To say that the sound produces an electrochemical effect in the auditory cells is, in my opinion, merely giving a name to an unknown quantity. If we knew what was meant by an electro-

chemical effect I might be able to agree or disagree with the proposed solution.

I believe I demonstrated that all that was necessary to cut down the resonant response in a resonator was to change the weight of the contained gas and indicated there was nothing to see. It is accordingly not necessary to believe we must visualize the adaptations to audition and perhaps we must hear them to understand them.

So far as my own theory of hearing is concerned, I cannot say that I really have one. My guess is that there is a peripheral analysis on the part of the auditory cells, and that discrete frequencies are resolved serially arranged from base to tip with highest frequencies at the base. I have no quarrel with either the Adrian explanation of the Weaver and Bray experiments on the basis of a microphonic action of the cochlea or with the explanation suggested by Weaver and Bray that the action currents in the auditory nerve reproduce the frequencies found in a composite source. So far as the mechanics of the system is concerned, both may be expressions of a similar effect. The latest experiments on turtles indicate that they do not hear sounds above 1000 Hertz, which conforms with the proposal that a progressive development takes place toward the high frequency resolution as the form evolves.

Sound vibrations are spoken of as waves because they may be visualized as transverse displacements rather than as longitudinal displacements. The "to and fro" swing in the molecules in reference to the wave length is readily shown in a transverse wave form. The sound advances something like a soap bubble distends.

Dr. Sonnenschein indicates it would be a good idea to standardize the fork for bone conduction and make it unnecessary to include the observer in the test. This is quite correct, provided the standardization on normal individuals is done under conditions of silence, because otherwise the readings will all be too low due to the masking of adventitious noises. Tests on Rinne negative cases with normal perception apparatus do not show a prolonged bone conduction. I am not concerned with the practical application of the test so long as we agree that a prolonged bone con-

duction does not occur except in cases with normal perception apparatus and with functional sound conduction system under conditions where the drum membrane is loaded or the external canal occluded.

DR. SONNENSCHN: In the clinical application, even if the premises are wrong, you have your criterion of comparison.

DR. POHLMAN: We are dealing with the mechanics, not with somebody's conception of the results obtained under unsatisfactory conditions of test. I have agreed that a conduction deafened individual with even depression in acuity throughout the audible range will show relatively greater disability at the low frequency end of the audible range. This, as I explained, was a function of the internal ear and was not dependent on the mechanics of conduction. I do not agree that in frank conduction deafness the low end shows greater losses in sensitivity than the high end of the audible range.

The theories of hearing, as I have tried to indicate, are nothing more than darns on the same old sock, and what we need is a new sock. Dr. Holinger objects to my attempt at wiping out all theories and starting in all over again. The tectorial membrane, as Dr. Shambaugh has demonstrated, is applied to the ciliae of the auditory cells just as the otolithic membrane is applied to the cells of the vestibular apparatus. If the cochlear apparatus were developed on the fundamental pattern of the elements of the otic vesicle, there is no reason why it might not take on the pattern of the vestibular system without definite function for either the tectorial membrane or the ciliae. Ciliae have persisted elsewhere without function. Structures do not disappear in the body merely because they are not used, but because their persistence may in some way interfere with selection. We have on the abdominal wall a muscle, the pyramidalis, which has only one function and that is to open the marsupium. People are just as fit to survive without a marsupium, and the persistence of the muscle may be accounted for on the basis that it does not interfere with survival.

Dr. Holinger has referred to instances in the human being with occlusion of the round window. He is probably referring to the two cases, reported by Oppikofer, where the niche was entirely

filled with fatty tissue. While the tests made on acuity by Oppikofer are not entirely satisfactory from an audiometric standpoint it is worth noting that he reported the two cases as having normal air acuity.

The experiments of Hughson and Crowe have suggested to them the function of the round window as a shock absorber, although no explanation of how this effect might be produced is given. It appears that the round window may only act as a shock absorber by having its vibrations in phase with those of the oval window. Under this condition the action would be to suppress transverse vibrations at the basilar membrane. However, the evidence is that a cotton plug against the round window affects the labyrinth through its vibrations, not its pressure, and if transverse vibrations are demanded in the cochlear duct for audition, then the individual should hear less well with the prosthesis in place. The evidence shows an increased acuity.

In answer to the question on the model reactions. Everyone knows that the size, viscosity of liquid, caliber of the tube, etc., influence the result. The pattern developed in the apparatus mentioned was apparently not influenced by the length of the column of liquid, and a pattern was developed only when the oscillations were from two to three to the second. Even at five to the second the pattern disappeared. We must also not forget that the air amplitude in the external auditory canal at minimum audition is a small fraction of a millionth of a millimeter, and the chances are that there is considerable loss in amplitude as the displacement is transmitted to the liquid of the labyrinth. It may be that this displacement must be expressed in terms of hundredths millions of millimeters. We are dealing with measurements which split the size of a molecule. The displacements probably pass through everything: basilar membrane, tectorial membrane, auditory cells and even the bony wall of the container. If Dr. Holinger believes displacements of this order can have an effect in the interaction of the cilia and the tectorial membrane it is his privilege, just as it is my privilege not to believe this possible.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL
SOCIETY.

Meeting Held on Monday, November 2, 1931.

THE PRESIDENT, DR. HOWARD C. BALLENGER, IN THE CHAIR.

CLINICAL PROGRAM.

Carcinoma of Larynx.

DR. ALFRED LEWY.

This patient had a squamous cell carcinoma involving both sides of the larynx, but intrinsic. A laryngectomy was done under avertin, supplemented by local anesthesia, which was very successful, except for severe paroxysms of coughing, during the separation of the larynx from the esophagus, which could not be controlled by local applications of cocain, and the esophagus was buttonholed during the dissection. This was repaired and healing was uneventful.

The important thing about this case was that after healing was complete the patient was referred to Dr. Leo Kallen for training in the esophageal voice. Dr. Kallen will discuss that phase. The man has made remarkable progress in eight weeks of training.

Rehabilitation of the Voice in Laryngectomized Patients

DR. M. REESE GUTTMAN.

These two men, aged 52 and 58, respectively, were operated upon by Dr. Beck and myself, one of them three and one-half years ago. Both are entirely healed and apparently there has been no recurrence of the malignancy. These patients are shown to demonstrate a few points that might be of interest in regard to subsequent voice production after the larynx has been removed. As is well known, there has been a large number of artificial larynges made, but, unfortunately, many patients are either unwilling or unable to learn to properly use them.

As far as the artificial larynges themselves are concerned, and aside from the fact that most patients do not take kindly to them, there comes in the question of their adequacy. None of the arti-

ficial larynges that we have tried has been very successful. In the Mackenty type, the vibrating rubber gets wet from the tracheal secretions and refuses to function. In the McKesson type, there is a great deal of difficulty in properly adjusting the vibrating reed. This adjustment fails every little while, and the patients also have a great deal of difficulty in learning to use the instrument. As with patients that have lost other members of the body, such as the nose and ear, they would rather have some substance that comes from themselves rather than of foreign nature. It is a well known fact that a patient without a nose or ear would rather have one built up of his own tissue than wear any of the artificial prostheses that are being made for this purpose, and apparently the same peculiar psychologic outlook holds for the laryngectomized patients. As a rule, they do not take kindly to the artificial larynx.

This brings us to the question of re-education of the voice. We have found this to be most satisfactory, but here, too, difficulties are met. Roughly, our patients can be divided into two classes, those that will talk and those that will not. Those that will talk apparently have the anatomic parts that are necessary to carry on conversation, and by that we mean they are able to swallow air into the esophagus and then belch it forth. They also have a well functioning cricopharyngeus, which apparently acts as a vicarious larynx. The tone produced by the air being expelled from the esophagus past the partially approximated cricopharyngeus can then be modulated by the tongue, palate and lips into fairly audible speech. In fact, a few cases of this kind have been reported in which the patient not only is able to speak but also can be made to sing. This one man (indicating), as you can now hear, has a fairly competent voice that is audible over a fair distance. He has taught himself, as he has the requisites of underlying anatomy in order to be able to produce tone. If this man had been under the care of a speech instructor he would probably have made a great deal faster progress. This man is fairly representative of the type that has a fair voice.

In this other patient, who has had his laryngectomy four and one-half years, we cannot get him to talk. Even under instruction he has been unable to learn, and a careful résumé of the case

will give us some idea as to why this is so. In the first place, a review of the operation will disclose that we resected the upper portion of the esophagus, including the cricopharyngeus, and if you will examine him you will notice that the esophageal introitus is wide open and relaxed and is filled with a frothy fluid that collects in the esophagus. This man is unable to approximate the walls of the esophagus in order to form a vicarious larynx, due to the fact that the cricopharyngeus muscle has been removed at the time of the operation. For that reason this vicarious larynx cannot be produced. However, one might produce a vicarious larynx by the use of the back of the tongue against the posterior pharyngeal wall or by the use of the palatal arches or the palate itself. Another factor may be present in this case, namely, this man's helpless attitude towards life. Apparently he does not wish to learn to talk.

A third patient, who disappointed us this evening, has a decidedly good voice. He is able to make himself heard across the extent of this room, and he produced this in a very peculiar way. This man performed an operation upon himself. He studied his condition and one day went to the trouble of heating up an ice pick and passing it from the fistula of the trachea in the neck up through the hypopharynx towards the base of the tongue. As the result of passing this instrument, a small fistula occurred and he is now able to place his thumb over the tracheal opening in the neck and then force air from his lungs along this small fistula back into the mouth. The air passing through this very narrow track forms a tone, which is then modulated in the pharynx and mouth in order to produce speech. Taking this remarkable example as a clue, Dr. Beck and myself have evolved an operation by which a needle is connected to a diathermy apparatus and is passed from the tracheal opening into the neck through the hypopharynx to the back of the tongue, staying well in the midline and coagulating a small track which later on will fistulate. This becomes lined with epithelium and remains more or less patent, and by placing the thumb over the tracheal opening into the neck the patient is able to force air along this fistulous track and produces a tone that can be modulated into voice production.

Discussion of Vocal Mechanism in the Laryngectomized Patient.

DR. LEO KALLEN.

The case just presented to you by Dr. Lewy has, I am sure, by a happy circumstance, been so prepared surgically as to offer anatomic conditions properly suited for the development of a so-called "esophageal voice." This patient came to me less than two months ago, and by diligent effort and intelligent application has already attained sufficient mastery over the mechanism involved as to be able to engage in a fair degree of comprehensible conversation. In order to make more clear the manner in which the esophageal voice mechanism works I beg your indulgence while I reiterate a few fundamental principles more or less familiar to all of you.

The normal production of voice and speech depends upon a coordinated mechanism consisting of three chief factors, viz., the lungs, the larynx and the articulation musculature. The manner of their control by the central apparatus does not concern us here. The sound engendered by the expiratory current of breath at the level of the vocal cords, as they are thrown into vibration by it, undergoes all sorts of modification in the parts superposed upon the larynx, namely, the resonance cavities of the pharynx, mouth and nose. Quick, agile variations in the shape and size of this resonator, as it might be called, give rise to the formation of the various consonantic and vowel values which we synthesize into meaning and call language. For a proper effect in speech, the coordinate function of the lungs, larynx and articulation apparatus must be executed flawlessly. That the slightest disturbance in any one of the three interferes with such an effect is a matter of daily experience.

Now what are the conditions in the laryngectomized? Not only is the vocal apparatus gone, but obviously the bellows function of the lungs is no longer present, a disconnection between the upper and lower respiratory tract having been effected by the operation. Thus the lungs can no longer function for phonic respiration. In the laryngectomized, respiration is reduced to vital function only. All that remains of the speech function complex is the articulation factor, and then only if it had not been

involved in the disease process and had thus been left intact by the operation.

It is thus clear that vocal therapeutic measures in the laryngectomized aim at the development and the ultimate establishment of a vicarious reservoir for air that may be subjected to compression so that some anatomic structure capable of functioning as a vicarious glottis may be activated by it.

The problem thus, first and foremost, revolves itself around the location of the anatomic structures offering possibilities for the development of a vicarious reservoir for breath, a vicarious "air chamber," as it might be called. Now it is reasonable to assume that the source of phonic breath must occur within a space closed off on all sides, in order to make possible the compression of such breath, which will then escape at a point of least resistance, namely, at that point where the vicarious glottis has been formed. Were this space not so closed off, the compression of its gaseous content would be quite impossible, and as a consequence neither could the formation of a vicarious glottis take place. One of the first steps in vocal gymnastic therapy is the accumulation of air within such a space, in as great quantity as is possible. It is obvious that the only possibility for the occurrence of such a space is somewhere in the upper alimentary tract between the levels of the mouth and the stomach.

As for the location of the vicarious glottis, there are many anatomic structures that can well serve the purpose. Any structure or structures capable of forming a closure or a narrow slit which can be thrown into vibration by compression and propulsion of air from below will do. Thus the dorsum of the tongue and the tensed velum palatini, the base of the tongue and the posterior wall of the pharynx, the two posterior pillars of the fauces, the inferior constrictor of the pharynx, the epiglottis and the two lateral pharyngeal bands, and lastly, but in my opinion most important, the cricopharyngeus muscle, alone or in combination, as the case may be, may all develop into vicarious glotti. The development of one in preference to another depends on several factors, such as the location of the vicarious air chamber, the new anatomic relations as established by the operation, individual architectonic variations, etc.

Before going more fully into the mechanism of esophageal voice, as exhibited by the case under discussion, I should like to say a few words about the two laryngectomized cases presented by Dr. Guttman. He has shown you one case in which speech developed spontaneously some time after the operation, which was performed two and a half years ago. The acoustic qualities of the voice in this case are typical of a pharyngeal mechanism, the pseudoglottis being somewhere in the pharynx and activated by a source of air from below. During the process of speech you can see the patient execute all sorts of oral and pharyngeal movements, but chiefly one with which he appears to be sipping in air. The cavity caused by the extirpation of the larynx and the hypopharynx is thus filled with air. This air, compressed by means of the muscular contraction of the parts that contain it, is thus expelled, and in such a way that it is directed toward and forced through a point of least resistance formed by a narrowing somewhere in the articulation apparatus above it sufficient to cause the production of sound. Very frequently this is the manner in which a "pseudovoice" is developed and maintained. After utilization of the air, its renewal is accomplished by the relaxation of those parts which contracted for its compression, the air rushing in as the result of negative pressure, as it would into any cavity which suddenly becomes larger. This inrush of air is equivalent to phonic inspiration. In addition, other movements are necessary, such as swallowing, sipping, etc. All the details of this process are very easily followed, for the inflation and collapse of the air chamber defines itself very clearly. Occasionally one sees a case (I have seen such a one recently) in which the upper part of the esophagus is involved in the function of an air chamber along with the cavity in the hypopharynx. In a great number of cases, on the other hand, it is the esophagus alone that functions for that purpose, the whole act of phonation being enacted within it, which brings us directly back to our own case. The act of speech here is ushered in by contractions of the esophagus which begin at the lower end of approximately its upper third, or even a bit lower, progressing proximally in a spiral or serpentine manner, as was first graphically demonstrated by Seemann. In the beginning of the therapy, this patient effected

the opening of the esophagus in a very explosive manner, but now that he has had a little practice, the whole process proceeds much more slowly and more purposefully, making it possible for much more air to reach the esophagus. In order to clearly understand this mechanism, a brief description of a very interesting phenomenon occurring in the esophagus and associated with the act of phonation is necessary. The dominant pressure within the esophagus is always a negative one except under conditions of forced expiratory activity, as may occur during screaming, coughing, etc. Motor manifestations may be observed in the esophagus during vicarious phonation. They are represented by synergic contractions consisting of a greater or lesser degree of tight, corset-like constriction of the esophageal lumen in the region of the esophageal mouth and hiatus. These constrictions are chiefly in the horizontal plane and represent genuine changes of an active nature which are to be construed as physiologic in character. This phenomenon evidently occurs where there is transverse striped musculature, receiving impulses conducted along fibers given off by the vagus, superior laryngeal and recurrent nerves.

These are genuinely active movements intrinsic to the esophagus and not incident to movements of respiration. The fact that the recurrent laryngeal nerves gives off branches, some of which, the so-called esophageal branches of the recurrent nerve, are distributed to the esophagus as well as the larynx, makes this supposition very plausible indeed, and is one of the very important reasons why the cricopharyngeus muscle should be preserved as much as possible during the operation. It is thus not unreasonable to suppose that this phonation phenomenon is a physiologic fact, which, normally represented by a latent potentiality, is stimulated to active kinesis in those very cases in which the larynx is wanting. In some cases in which the esophagus is the vicarious air chamber these contractions exert their force for the purpose of compressing the air, while in other cases the constriction which they cause can well be utilized as a vicarious glottis, when the source of air occurs below it. It is thus clear that the anatomic and physiologic conditions of the esophagus perfectly meet the requirements for the production and development of a

vicarious voice. The laryngectomized subject has to learn the effective aspiration of air into the esophagus which ordinarily contains a negative pressure, by means of conscious sensibilities located in the parts involved. In the beginning the process is accompanied by a belch-like sound which is caused by the friction of the air current and the dishesion of the esophageal walls. The moment the aspiration sound occurs, tension of the oral floor can be felt, while the region of the larynx executes a jerk-like movement upwards and forwards through the contraction of the thyrohyoid muscles. The force represented by this upward and forward pull is simultaneously distributed to the cricopharyngeus muscle which forms the anatomic substratum of the esophageal orifice. This is the manner in which the tonic closure of this muscle is relaxed. Simultaneous with the described movement, a quick, short inspiration, effected by the execution of a jerk-like contraction of the diaphragm, causes an inrush of air into the esophageal vacuum with the production of the described acoustic effect. The striking point about this case, however, is that not only can the subject speak during the expiratory phase of this vicarious phonic respiration, but he has also been taught to transform the sound engendered by the act of respiration into articulate value as well. He is thus able to articulate the aspirate sound and the sound incident to the process of expulsion. This in time will make for a remarkable degree of fluency. The only other case of a "two-way" speech, so to speak, as reported in the literature, is one described by Emil Fröschels many years ago. His case, however, developed the mechanism spontaneously.

Just a word in regard to Dr. Guttman's second case. It is obvious that this patient has no tonal voice. His manner of speech is represented by a confluence of oral postures, some of which effect the pronunciation of voiceless consonants, but no more. Such a process is referred to as "pseudo-whispered" speech. This, however, is a misnomer, for the act of whispering depends upon the larynx fully as much as does tonal voice, the normal whisper being due to friction of the expired breath through a partially open glottis. The effect of whispering in this laryngectomized subject results from a compression of air contained within the oral cavity when articulation functions for the production of

nonvocal consonants of explosive or fricative character of short duration. There is no pseudoglottis in these cases. The vowel, which is always sustained, is represented merely by the oral posture involved, since there is no source of air for its sustenance. This patient is a wonderful example of phonetic neglect after operation. There is no doubt that he could have developed a voice with the proper training and instruction. Here he is, five years after the operation, and yet not adjusted to the new conditions as he should be. This manner of speech occurs spontaneously soon after the operation, and although more or less audible, is under the most favorable circumstances comprehensible only when the listener is at the peak of attention or at very close range. A patient who becomes habituated to the pseudo-whispered voice has established a condition which makes the further mastery of a laryngeal speech by vocal method extremely difficult. He gets conditioned to it, even by a short period of practice, and finds himself confronted with great obstacles at the beginning of vocal gymnastic therapy, the new mechanism gaining a foothold only very poorly or not at all. It is thus of the utmost importance that the patient, after successful operation, be warned most vehemently against the danger of resorting to this manner of communication and to make every effort of consciously avoiding it. It is far better for him to communicate with pencil and paper than by this means until such time as vocal gymnastic therapy is undertaken.

In closing, I would like to emphatically stress the necessity for the surgeon to be interested in preparing the patient as favorably as possible for the phonetician. This should be an important factor in his technic. He should spare as much tissue as is safely possible. In his desire to be absolutely sure he will often remove demonstrably sound tissue, such as muscular tags, folds of mucous membrane, etc. The removal of such tissue should be limited to cases only in which doubt as to the health of the tissue makes it imperative. Every fold of mucous membrane, every favorably placed cicatricial band, every muscular remnant may serve as the basis for the development of a pseudoglottis. What they are or where they should be placed cannot be said in advance. The vicarious expiratory current of breath selects, on the physical

principle of efficiency and mechanical advantage, that structure which in resistance to it, will best meet the mechanical principles involved—in other words, that structure making most possible the maximal production of useful sound with the minimal application of force. In this way, many a striking case of pseudo-voice owes its development to the happy hand of the surgeon. The question thus arises as to the possibility of surgically predetermining and inserting a pseudoglottis. It has been suggested that the transplantation of two folds of mucous membrane or muscular strips in the meso or hypopharynx might serve as the structure which later develops into the pseudoglottis. The position or direction of these elements is not a matter of importance. What counts is their ability to vibrate. Only long experience and eternal vigilance along these lines can teach us ultimately how to improve and perfect the formation of a pseudoglottis. The cricopharyngeus, above all, must be spared. The integrity of its innervation must be preserved to the greatest possible extent. Protection of the sternohyoid and thyrohyoid muscles is also very important, for these muscles pull the laryngeal region upwards and forwards, lifting it away from the posterior wall of the pharynx whereby the tonic closure of the cricopharyngeus is relaxed, a very important factor in the training of esophageal speech. As a final surgical prerequisite, from a phonetic point of view, these muscles should be sewed to the anterior wall of the pharynx, approximately at the level of the mouth of the esophagus, for the purpose of facilitating the mechanism of aspiration.

Carcinoma of Throat.

ROBERT SONNENSCHIEIN, M. D., AND SAMUEL J. PEARLMAN, M. D.

This patient, a physician, aged 58, was first seen on May 7, 1931. He complained of periodic hoarseness for the past twenty years. The last attack had occurred during the past five months and was associated with some discomfort in speaking and swallowing, but no cough. The Wassermann reaction one week before had been negative.

Examination of the pharynx showed flat, submerged tonsils with caseopurulent material in the crypts. The vocal cords moved freely but a mass was seen involving the anterior three-fourths of the left cord. No glands were palpable in the neck.

A roentgenogram of the chest on May 11, 1931 (Brams) revealed tuberculous infiltration of the left apex, a calcified nodule of the right apex and bilateral inactive tuberculosis.

A biopsy performed at Michael Reese Hospital, on May 19, 1931, was reported squamous cell carcinoma of the left cord.

On May 19, 1931, under cocain anesthesia, with the Hasslinger forcep, tissue was removed from the left cord.

On May 27, 1931, under amytal-morphin, with novocain blocking, an incision was made from the thyroid to the sternum. The second and third tracheal rings were cut and a large tube inserted. The thyroid was split to the right of the median line. The left wing was resected subperichondrially and the left membranous larynx was removed beyond the tumor tissue. A few arm silk sutures were inserted and gauze dressings applied.

This second patient, Mr. H. P. H., aged 61, was seen on May 3, 1929. He complained of hoarseness for eight months and of some pain in the neck on the left side. There was no dysphagia. He had consulted Dr. Mackenty in New York, who diagnosed carcinoma of the larynx.

Examination showed that the nasal septum deviated to the right. The tonsils were flat and inflamed. The right cord was polypoidal in the anterior two-thirds. The left cord was discolored, with an uneven edge, but moved freely. There was a rounded tumor-like swelling of the left false cord and a small gland could be palpated over the lower trachea.

On June 17, 1929, under cocain infiltration, a median incision was made from above the hyoid to the lower part of the trachea. The hyoid was cut with forceps, the muscle separated and the trachea exposed. Packing was placed below and the skin was closed with silk down to the packed area.

On June 21, 1929, under cocain anesthesia, the upper tracheal rings were incised and a tube inserted.

On June 24, 1929, under morphin-scopolamin, cocain and novocain, a thyrotomy was performed, with diathermy applied to the tumor tissue on the left side. The tube was removed about July 24, 1929.

On August 2, 1929, the left side of the larynx appeared smooth, his voice was fair and the neck wound was healed.

On March 11, 1930, the left false cord was large but smooth.

I will now show you some lantern slides of a case of osteoma of the maxillary sinus and of a case of diverticulum of the esophagus.

Carcinoma of the Antrum.

T. C. GALLOWAY, M. D.

For carcinomatous lesions of the antrum it seems to have been shown that nothing is so successful as surgical diathermy. My three patients were presented this evening to demonstrate the fine healing that occurs, with a relatively small amount of mutilation.

The first patient had a lesion the size of a lemon, which involved the wall of the nose, the upper lip and extended into the antrum. It was reported as an epidermoid carcinoma. He had also four small typical basal cell carcinomas, proven by biopsies, in the adjacent atrophic area. These lesions were destroyed by the superficial desiccating current and the large lesion by electro-coagulation. That case is now one year old, and plastic repair by the pedicled flap shown is under way by Dr. Joseph Schaeffer.

The second patient had a lesion that involved the naso-antral wall and the inferior and middle turbinate, going into the mouth and involving the orbit and the ethmoids, filling completely the maxillary sinus. We went through the skin and destroyed the lesion and overlying tissues. I think we should use the external approach in all such cases, in order to see and destroy the growth completely. If we try to carry out a cosmetic technic we often will not obtain satisfactory results. This man has a large defect remaining from the operation, but it is smooth and completely healed. If a local recurrence appears in such a lesion it can be picked up by biopsy within a few weeks. This cannot be done after surgical or radium therapy. The last diathermy was three years ago. The defect in this case has been repaired by a flap from the shoulder by Dr. Schaeffer with splendid results.

The third man had a lesion over the canine fossa. On lifting the upper lip we could see at the buccal angle a rounded swelling which went up to the orbit, and there was a smooth rounded swelling through and into the nose. Five biopsies were performed before the laboratory reported that the growth was an

epulis and not simple inflammatory tissue. It is a rather unusual manifestation of this tumor. The case was attacked repeatedly by diathermy and with radium and responded well to this treatment.

The film shown is of a patient who was struck by a bullet which carried with it the upper left central incisor, ranged the length of the tongue and buried itself against the cervical vertebrae. She was desperately ill when seen three days later and apparently in surgical shock, although the real trouble proved to be dehydration and fusospirochetal infection, which had previously been overlooked because of emphasis on shock. With intravenous dextrose, Ringer's and arsenicals the whole picture changed in twenty-four hours. The bullet was removed under the excellent deep illumination of the Good light. The importance of preventing dehydration is to be emphasized in all cases where obstruction makes swallowing difficult, notably infections, foreign bodies, malignancies and injuries.

Gumma of the Septum.

SAMUEL SALINGER, M. D.

Case 1.—This woman, aged 35, was about to have a submucous resection of the septum done for what appeared to be a deviation of the quadrangular cartilage. Upon attempting to inject it with novocain I found that the needle entered into what seemed to be a soft cavity. I deferred operation and took a Wassermann test, which came back plus four. She now has a typical gumma of this region and antisyphilitic therapy is being carried out.

A few years ago, in a similar case, I was not so suspicious but went ahead with the operation. The result was a large perforation and saddle nose.

Temporal Lobe Abscess.

Case 2.—A boy, aged 5, had a chronic running ear for four years with acute exacerbations. A few days prior to admission there was a sudden cessation of the discharge, severe headache, projectile vomiting. Temperature 98.6° F., pulse 64, spinal fluid under pressure, Pandy positive and forty-five cells. There was a bilateral choked disc, more marked on the right side. On the basis of these findings we opened the mastoid, which was sclero-

tic, and found the antrum filled with cholesteatoma and granulations. The tegmen was removed, the dura of the middle fossa found bulging and tense. It was opened and a brain searcher introduced upward for about 2 cm., where resistance was encountered. On penetrating this point there was a gush of foul smelling pus. The abscess cavity seemed to have a definite capsule and was drained with a rubber tube for about two weeks. During this time saline and peroxid irrigations were employed, alternating with suction.

The boy seemed to be perfectly well after two months, although the choked disc is still apparent and there is a slight tendency to external rectus paresis on the opposite side, which has been present since the operation. There are no other symptoms.

In the treatment of these cases, as in other brain abscesses, we have found it of advantage to sit the patient up immediately after the operation and to keep the head turned away from the side of the lesion. This has a tendency to keep the abscess cavity from collapsing.

I don't think it makes much difference as to what method of drainage is employed so long as the drainage is unobstructed. The important facts are that if the abscess is well capsulated and the virulence of the organism well attenuated the patient has a good chance for recovery.

In all other cases, as is generally known, the prognosis is very bad.

Carcinoma of Larynx—Laryngectomy.

Case 3.—A man, aged 58, had an extensive carcinoma of the larynx, which was beginning to be extrinsic, but nevertheless seemed favorable for operation. We did a one-stage laryngectomy without preliminary tracheotomy. We found on opening the neck a carcinoma had perforated the thyroid cartilage on the right side, a secondary infection had developed, and there was an abscess in the region of the perforation extending backward to the posterior border of the cartilage. Despite these unfavorable findings we went ahead with the operation and removed the larynx with the epiglottis and considerable of the muscle tissue in front of the larynx. The patient made an uneventful recovery

and thus far has shown no signs of recurrence. He is receiving deep X-ray therapy as prophylaxis.

The pathologist's report on the specimen was epidermoid carcinoma.

Cerebellopons Angle Lesion Syndrome.

DANIEL B. HAYDEN, M. D.

Case 1.—This man, aged 45, was examined in the clinic on October 6, 1931. He stated that he became suddenly deafened in the left ear fifteen months ago, the onset of the deafness being associated with marked dizziness and pulsating tinnitus. For the first three days the dizziness was quite marked, he noticed that when walking he had a marked tendency to deviate to the left and he was unable to work. At no time was there pain in the left ear. There was no history of any previous ear trouble other than recurring attacks of eczema.

When examined the tympanic membranes were found to be normal. The left maxillary sinus was cloudy. This was irrigated and a small mass of flocculent pus was removed. There had been a submucous resection of the septum nasi, with resulting perforation. The tonsils were completely enucleated. The larynx was negative.

Hearing tests: Weber to right, Schwabach negative, Rinne, right positive, left absolutely negative. There was total deafness in the left ear for all tuning forks. There was no spontaneous nystagmus. Spontaneous pointing, the right and left hand touched. Rotation to the right ten times in twenty seconds elicited no response. Rotation to the left at the same rate elicited nystagmus normal in direction and amplitude, but of shortened duration, ten seconds.

Caloric tests: Cold water at 68° F., head in erect position. Stimulation of the right ear for five minutes produced no response. Tilting the head backward showed a normal response to left. Right, hand touched. Left, hand touched. Stimulation of the left ear for five minutes produced no response. Right, hand touched. Left, hand touched.

The audiogram showed hearing to be within normal limits for the right ear. There was total deafness in the left ear for all

tones except 1024 and 2048, which were perceived after 80° of amplification, but the patient volunteered the information that they were being heard in the right ear.

The blood and spinal fluid Wassermann reaction is negative.

The report of the neurologic examination, briefly, was: "There is no evidence at the present time of any angle tumor."

Here, then, is a case of total destruction of the function of the left cochlea and of the horizontal and vertical canals on the left and of the verticals of the right side. This is the so-called "cerebellopons angle lesion syndrome," indicating a lesion, whether exudative, infiltrative or neoplastic, of such a nature as to block afferent impulses of the entire homolateral eighth nerve and afferent impulses of the opposite vertical canal. It is possible, however, that we have here a patient whose labyrinthine responses are the first to be affected and that later well marked neurologic symptoms will appear.

Nasal Polyp (Fibromyxomatous).

Case 2.—This boy, aged 11, was first seen in June, 1931. He gave a history of nasal obstruction which had been present for the past three years, and of severe attacks of epistaxis during that period. Recently he had complained of pain over the left cheek and his parents had noticed swelling beneath the left eye.

Examination: Transillumination showed the right antrum clear, the left dark. Roentgenograms made at the Research Hospital showed the left antrum and ethmoid sinus dark, the other sinuses clear.

The right nasal passages were perfectly free, the left partially blocked by what appeared to be a large mucous polyp lying on the floor of the nose, filling the inferior meatus and apparently attached to the middle meatus. It was movable, firm, grayish and bled easily on manipulation. A large piece was removed, followed by a profuse hemorrhage.

The microscopic diagnosis was: Fibromyxomatous polyp of the nose.

In view of this report, further effort was made to remove the entire mass. It was not possible to enclose the entire mass in the loop of the snare, and on cutting through it the hemorrhage was so profuse that the nose had to be packed.

The microscopic diagnosis on this piece of tissue was: Hemangiomatous tissue.

Because of the severe bleeding, 25 mgs. of radium was applied in the left nostril for four hours on July 30. This seemed to slightly reduce the size of the mass, and six weeks later a further attempt was made to remove it, but while cocainizing about the middle meatus such a profuse hemorrhage occurred that only tight packing controlled it. On October 23rd, 12.5 mgs. of radium was applied in the left nostril for six hours.

At present the postnasal view shows the tissue completely blocking the left choanae and extending into the nasal pharynx, obstructing the view of the left pharyngeal opening of the tuba auditiva. A roentgenogram shows marked clouding of the left maxillary and ethmoid regions.

The right tympanic membrane is negative. The left shows marked retraction and slight injection of the superior portions. After inflation the tympanic membrane returns to normal position. The whispered voice, low tones, is heard at three meters and the higher tones at four meters in both ears. Big C is slightly shortened in each ear by air conduction. There is a shortened positive Rinne in the left ear.

Laryngofissure.

M. REESE GUTTMAN, M. D.

This man, aged 58, had a laryngofissure performed for carcinoma of the larynx by Dr. Beck and myself five and a half years ago and is presented to show some of the vagaries that a malignancy may at times exhibit. When first seen, in 1925, he complained of hoarseness, and examination of the larynx showed a small growth on the vocal cord. There were no palpable glands in the neck. The small size of the lesion and its situation within the larynx made us consider the case suitable for a laryngofissure, which was performed. The cord was removed, the patient recovered rapidly and was discharged from the hospital. Unfortunately, the neck wound refused to heal, due to the fact that prolific granulations continued to form in spite of all measures. After a number of weeks had passed, a biopsy was taken on the granulations in the neck and the report came back "squamous

cell carcinoma." He was then advised to have a laryngectomy, but he refused and instead went to the Mayo Clinic. There, also, a laryngectomy was advised and he again refused, and the patient then returned to Chicago, where he was under our care for a very short period of time. He then disappeared and when next heard of he was under Dr. Pearlman's care at the Michael Reese Dispensary. A few months later he appeared in the office with the entire larynx and cervical wound healed.

It appeared that Dr. Pearlman did nothing for him except observe him and have him dressed. Much to our surprise, the lesion has remained well during this period of five and a half years. There has been no evidence of any recurrence. The intralaryngeal picture shows a smooth fibrous mass at the site of the original vocal cord. We have biopsied this on three different occasions, the last one being about four weeks ago, and in each case the report has been returned negative.

Apparently this is another case of a spontaneous cure or regression of a malignancy.

Radio-Sensitive Malignancies About the Oral Pharynx.

M. REESE GUTTMAN, M. D.

This man, aged 62, was first seen by us about eighteen months ago, complaining of pain on deglutition. Examination at that time revealed a small neoplasm, about the size of a walnut, at the base of the tongue, and apparently also invading the tonsil. A biopsy was performed and the report was returned "transitional cell epithelioma." He was treated by the insertion of radon seeds and within a period of three weeks the lesion disappeared. There has been no recurrence for the past eighteen months.

This second patient was first seen by us about fourteen months ago. He was referred by his family physician, who found a tumor the size of a cherry at the base of the right tonsil. This entire tumor was removed and the histology showed it to be a lymphosarcoma. Under irradiation the base of the lesion, from which a biopsy had been taken, and was also shown to be lymphosarcoma, healed and apparently has remained well to date.

These two very interesting cases bring to mind a third case that was seen by us about six weeks ago. The patient had a large

mass in the back of the nose, which had been present for a period of about four months. The patient also exhibited large glands in the neck. A biopsy was returned "lympho-epithelioma." Knowing the highly radio-sensitive nature of this lesion, radon seeds were distributed throughout the nasopharyngeal mass and deep therapy was given to the gland in the neck. Within a week all evidences of the tumor within the nasopharynx and the gland in the neck had disappeared. Just what the future outcome of this patient will be is, of course, as yet problematical.

We are learning a great deal today from the histologic investigation by the men who are interested in radium. It is due to their efforts that we have been able to isolate such clinical entities as the lympho-epithelioma, the transitional cell epithelioma and Ewing's endothelial myeloma.

These three types of lesions are highly radio-sensitive, and they respond to even small doses of radium or deep X-ray therapy. However, radio-sensitivity must not be mistaken for curability. It is a peculiar fact that these lesions are highly malignant and that they very rapidly metastasize and cause death. Although they may respond promptly and dramatically to doses of radium or X-ray, yet the prognosis, due to the fact that metastases are early and widespread is, as a rule, poor.

Foreign Body in Right Bronchus.

EDWIN MCGINNIS, M. D.

Case 1.—A patient seen within the past few months had inhaled a glass ornamental jewel which had lodged in the right bronchus. This ornament was faceted and the dimensions were about one-fourth by three-fourths and it was one-fourth of an inch thick. It was rather ovoid in contour and was hard to hold onto with the ordinary grasping forceps. I finally had to put in as large a tube as the larynx would carry so as to get a sufficiently strong forceps down to it. This forceps had some fine teeth on both blades and with this I was able to get a firm hold on the object. Removal was accomplished and the patient made an uneventful recovery.

Case 2.—A boy, aged 3, had been playing on the beach and was suddenly taken with a coughing spell. A roentgenogram

showed a faint shadow in the right bronchus, and the breath sounds on the right side were reduced. When we got the child on the operating table, however, we found the object in the left main bronchus. We discovered on removing the object that it was a small smooth pebble. The only forceps which would hold this was a basket-shaped one patterned after the Bruening type.

Case 3.—This was a boy, aged 4, with an open safety pin in the right bronchus. This pin had been in place for about ten days and one attempt had been made at removal which was unsuccessful. I was unable to close the pin, but got it out through the tube by grasping the guard. This child made an uneventful recovery.

Case 4.—A two-year-old child had inhaled a kernel of corn, which was located in the lower end of the trachea. He had been taken to a local hospital and an attempt at removal failed. He was then brought down to Chicago and I saw him the following day. The resident on the pediatric service and my own interne, after examination of the chest, said there was nothing in the air passages. On asking the child to cough, one could hear the object moving up and down in the trachea. I removed this, which proved to be a kernel of yellow dent corn, in less than a minute. I instructed the interne on duty to call me immediately if the youngster showed any untoward symptoms during the night, but unfortunately this was not done and the child died early the next morning. On investigation I found that the child had had a little difficulty in breathing and the resident decided to do an intubation. Unfortunately he had not had much experience and the youngster immediately became cyanotic, and even though an emergency tracheotomy was done the child died. A postmortem was not done, so we have to guess as to why this child died. One supposition is that he may have developed a pneumonia, and another one is that the thick tracheobronchial secretions may have caused death by drowning.

I am always a little bit on the *qui vive* when I have vegetable material in the tracheobronchial tree.

Auto-Tympanoscopy.

AUSTIN A. HAYDEN, M. D.

It is sometimes of interest for a patient to see his own ear drum, and this can easily be done by using an ordinary ear specu-

lum and putting a mirror on the back of it. By using a second mirror it is quite easy to see around the corner if the mirror is properly angulated, and the patient can see his own ear drum. This is particularly valuable in the presence of perforated ear drums, for it makes the patients realize that they should not put water in the ear, and it also has some influence on the proper toilet of ears. I have given this procedure the name of autotympanoscopy and shall be glad to demonstrate it to any who are interested.

Laryngectomy for Carcinoma.

HARRY L. POLLOCK, M. D.

This man, aged 62 had a laryngectomy performed fifteen months ago. He had a history of hoarseness of five years' duration and had been treated by his family physician until two weeks before I saw him. I operated in August, 1930. He made an uneventful recovery and remained well for twelve months, when he developed difficulty in swallowing. There was always some induration on the right side of the wound. Two biopsies were performed, with three pieces of tissue removed each time, all of which were reported scar tissue and no carcinoma. The difficulty in swallowing increased until he could take nothing but fluids. We put him under the fluoroscope and found a circular constriction just above the tracheotomy opening. We tried to pass an esophagoscope, but even the smallest bougie would not enter. It was very difficult to manage the patient, so we decided to open the esophagus, cut through the stricture and dilate it. This was done, and by passing dilators up and down and keeping the region open by means of a tracheotomy tube kept in place for twenty-four hours at a time, at the end of four or five weeks we had dilatation as large as the normal esophagus. We intended to close the wound by a plastic procedure, when the patient developed a small abscess in the skin, so this has been postponed.

I think we do not see many cases in which after a year the esophagus becomes so constricted without any recurrence of the malignancy. In this case a biopsy at present shows no evidence of recurrence.

Discussing laryngofissure, I do not believe we should do this operation at one sitting. Dr. Tucker has stated that in about 40

per cent he has to do a second operation in these cases to remove the granulation tissue. My habit is to do a tracheotomy and then pack the patient for about a week solid. We never get any granulation tissue and the wound heals well. In one patient operated upon two years ago we split the hyoid bone and packed him, and in about three to four months there was reformation of the scar tissue which resembled the normal cord.

Continuous Rhythmic Movements of the Palate, Pharynx and Larynx.

NORMAN LESHIN, M. D.

Continuous rhythmic movements of the palate, pharynx and larynx occurring synchronously are of considerable interest because they are comparatively rare, and they signify definite evidence of organic brain disease. In reviewing the literature up to 1930, Dr. Stone and I have found only twenty-seven cases, including our own, showing these movements. Since then Jelinek and Sachs, in the Czech Medical Journal, January, 1930, and Childray and Parker, in the Archives of Otolaryngology, August, 1930, each report an additional case, bringing the total number to twenty-nine. These cases should not be confused with those having isolated movements of the palate and pharynx which are more common and may be functional in etiology.

Twenty-four of the cases reviewed showed definite evidence of organic brain disease. Nineteen cases revealed positive evidence of cerebellar dysfunction, fourteen had findings of cerebral arteriosclerosis and three of bulbar palsy. Only six cases included postmortem reports. All these revealed cerebellar as well as cerebral pathology. In three of those reported by Klien, left sided movements showed a left cerebellar disease, right sided movements right cerebellar pathology and bilateral movements bilateral cerebellar involvement. From this evidence we feel justified in assuming a central brain lesion when these synchronous rhythmic movements are present.

The common characteristics of these movements are that they involve the palate, pharynx and larynx. They are constant, synchronous, gross and oscillatory, having two phases, a rapid and slow, bearing no relation to the pulse or respiration. They persist under anesthesia and are usually not influenced by extraneous

stimulation or the physiologic functions, nor do they interfere with these actions. The movements always occupy the same plane and direction and have a constant rhythm, their rate being usually between 120 and 180 per minute, rarely above or below these points. The patients are generally unaware of their presence. There is usually no evidence of local paralysis or disease. The movements of the larynx are generally bilateral, whereas the movements of the palate and pharynx may be either unilateral or bilateral. In only five cases were the movements of the larynx unilateral, associated with unilateral contractions of the palate and pharynx on the same side.

Case report: A male, aged 42, gave a history of progressive weakness of the left arm and left leg for the past five years. The essential neurologic findings were left hemiparesis, including the face and tongue, with postplegic hyperkinetic movements of the left upper extremity. The left lower extremity was spastic and ataxic with a left inexhaustible ankle clonus and positive Babinski. The deep reflexes were bilaterally brisk, while the abdominal and cremasteric were absent on the left side. There was a spontaneous nystagmus of the first degree when looking in either direction and a vertical nystagmus when looking upward. The entire posterior pharyngeal wall was in a continuous to-and-fro movement to the left at a rate of 132 per minute. The left posterior faucial pillar was also in rhythmic motion, moving towards the midline and back again synchronous with that of the pharynx. Slight movements were transmitted to the palate, especially on the left side, which seemed to rise and fall. The uvula showed rotary backward and upward movement. Examination of the larynx revealed both vocal cords and arytenoids in similar rhythmic motion. The true vocal cords and arytenoids approached each other and then returned, occupying approximately one-third their normal excursions, never meeting in the midline. The right cord moved more than the left. The adduction was quicker than abduction, and the rate here also was 132 per minute. The movements ceased during phonation. Slight synchronous movements were also present in the false cords and both aryepiglottic folds, especially the right. All the movements were synchronous at a rate of 132 per minute. The composite picture is a quick phase

composed of the rising of the palate and uvula, movement to the left of the posterior pharyngeal wall with adduction of the left posterior faucial pillar and both arytenoids and true vocal cords followed by a secondary slower return of these structures to their former positions. A diagnosis of multiple sclerosis was made.

The motion picture of these movements shows their characteristics very clearly. A complete review of all the cases reported showing these movements, including a detailed description of this case, will soon appear in the Archives of Neurology and Psychiatry.

DISCUSSION.

DR. ALFRED LEWY: This is the finest lot of motion pictures of the eyes, palate, larynx and pharynx I have ever seen. They are better than those made in some of the university laboratories with much more elaborate equipment. I would like to have Dr. Leshin tell us what kind of a camera he used, what lens, what aperture, what the illumination was and whether or not any special mirror was used.

DR. NORMAN LESHIN (closing): A large laryngeal mirror was used for photographing the movements of the larynx, the indirect image being photographed from the mirror. Two 500-watt lights were used for the exposure and a model A Eastman cinekodak was used with a telephoto lens and an S4 aperture. A reversal 16-millimeter panchromatic film was used.

ABSTRACT OF TRANSACTIONS OF THE AMERICAN
OTOLOGICAL SOCIETY.

SIXTY-FOURTH ANNUAL MEETING.

June 18th, 19th and 20th, 1931.

Briarcliff Lodge, Briarcliff Manor, New York.

ABSTRACTED BY DR. ROBERT SONNENSCHN, CHICAGO.

THURSDAY MORNING SESSION, JUNE 18, 1931.

The sixty-fourth annual meeting of the American Otological Society, Inc., convened at Briarcliff Lodge, Briarcliff, New York, at 9:00 a. m., Dr. D. Harold Walker of Boston, president of the Society, presiding.

Presidential Address. Otological Problems.

DR. D. HAROLD WALKER,

BOSTON.

Nearly all of the allied sciences must be understood and co-ordinated if the otologist of today is to enjoy to the fullest degree the study of the organ of hearing. He must realize that research and investigation should go "hand in hand" with clinical otology if he wishes to develop his individuality and power of imagination and to maintain a keen scientific intellect for years to come.

I do not wish to spend valuable time in a prolonged discussion of the many problems to be solved and the unknown fields to be discovered, but the recent researches of a few investigators make it imperative that a few of these lines of research be mentioned, and I would also like to suggest a few subjects to be considered by the Society for their future programs.

Three years ago Dr. Goldstein gave a most interesting paper on the "Organ of Hearing in Insects." Do you realize that only in one textbook on Otology is there any mention of the comparative anatomy of the ear? Perhaps every otologist remembers

perfectly the development of the ear from the lowest form to the highest, but I doubt it. I believe that a clear, concise, illustrated paper upon this subject would be most acceptable to the members of the Society. Again, how many of us have a clear conception of the nerve tracts; their distribution in the brain stem and to the cortex? It is difficult to remember the intricate system of fibers, especially in the medulla. I think we all would welcome such a contribution, especially if it were illustrated with charts.

A good deal has been written about the best method of attacking infection of the petrous pyramid; the last word has not been said. It is a very serious thing to attempt such a procedure, and only the most experienced operator, with a detailed knowledge of the anatomy, should have the courage to perform such an operation. Further work upon this subject would be most valuable. Recently a method of treating septic meningitis by injecting chemical agents into the arterial circulation has been suggested and practiced. Before such methods be strongly advised, a definite knowledge of the rate and extent of diffusion of substances from the blood stream through the choroid plexus to the spinal fluid should be determined by experiment, especially as to their bactericidal power, etc.

The spectacular experiments of Wever and Bray of Princeton last year may well be the beginning of the accumulation of scientific data, which will revolutionize the theories of hearing and enable us to attack the serious problems of deafness, with some chance of giving relief. Briefly, the experiment consisted of attaching an electrode to the auditory nerve of a cat, which led to a loudspeaker. Words and sentences spoken into the cat's ear could be heard given out of the loudspeaker. Adrian of Cambridge, England, doubted that the electrical stimulus traveled by the eighth nerve, but thought it was due to a dissemination through the temporal bone. Other observers have corroborated this experiment of Wever and Bray, so that their findings are probably correct. The future of this work will depend upon the co-operation of the physiologist and the otologist—all appreciating the necessity of observing chemical and physical reactions and their results.

Just think of the numerous theories of the functions of the drum, ossicles, eustachian tube and muscles which require either proving or disproving. We are fortunate in having a paper upon this program by one who has taken advantage of this experimental work. We await his results with interest. At the physiologic laboratory, at Harvard, several most elaborate and interesting experiments are planned for the autumn.

These subjects are only a few of the ones which need investigation. There has been a feeling among the profession, and the laity, too, that Otology has fallen behind its sister specialties and that it has not made any material advance. I hope this short address will tend to stimulate each and every otologist and instill enthusiasm and faith in the future of Otology. If we are not able to help by doing research work ourselves, then it is our duty to put our shoulders to the wheel and to aid in every possible way the investigator in the laboratory and elsewhere, who is patiently and quietly working in the dark, so that we may have light.

DR. WALKER: It is an honor and a privilege to welcome you here to this beautiful place on this beautiful morning. I will not delay this interesting program by my address, but it is necessary to open this, our sixty-fourth annual meeting, with a few words. I sincerely hope that when you leave you will speak of it as one of the most interesting and enjoyable meetings you ever had.

Report of the Committee on the Study of Progressive Deafness.

DR. ARTHUR B. DUEL,

NEW YORK.

I only wish to inform you that the fund for the investigation of otosclerosis is now functioning. We have in hand a fund which provides us with an income of \$21,900. It was announced that a total of \$500,000 was to be donated for this investigation, but of that \$56,000, which is pledged, is not in hand as yet, so we have not as much as we expect to have when the pledges are all in. However, we hope that the interest in the work being done

will stimulate some philanthropist who will double or treble the amount which we have to work with. One thing is certain, that we have an earnest group working toward a definite purpose, and this makes us hopeful that we may finally solve the problem.

Report of the Committee on the Study of Progressive Deafness.

DR. J. GORDON WILSON,

CHICAGO.

During the year May, 1930, to May, 1931, the investigations subsidized by your committee have been carried on at various laboratories on different aspects of the subject. It appears to your committee that the results have been most gratifying. A summary is here presented.

I. Anatomic and Embryologic.—Dr. Bast, at the University of Wisconsin, has continued the study of the development and ossification of the ear in man. To this he has added a study of the vascularization of the embryonic ear.

1. He has made serial sections of twelve ears.
2. He has built three models of human ears and two of monkey ears, to determine accurately the position, extent and relation of the ossification centers.
3. Three articles were published:
 - a. Ossification of the Otic Capsule in Human Fetuses. June, 1930.
 - b. A Comparative Study of the "Utriculo-endolymphatic Valve" in Some of the Common Mammals; September, 1930.
 - c. Blood Supply of the Otic Capsule of a 150 mm. (C. R.) human fetus. January, 1931.

In the report he says: "I have received no response to my appeal for fetuses from hard of hearing stock, and this fact is slowing up the projected work. In the hope of obtaining such material and of material from young children, I am laying plans to visit the superintendents of the children's institutions throughout our state some time this summer."

"During the past one-half year we have concentrated our attention on the ossification of that part of the otic capsule surrounding the semicircular canals. The cartilaginous capsule in this region has an abundant blood supply which seems to have no relation to the ossification process. We have made three models to study this and we have several more to make in order to have a complete series.

II. Dr. Crockett's report will be incorporated in a paper to be presented separately.

III. Hearing in Animals.—A method of testing adequately the hearing of animals is a prerequisite to experimental work in hearing. This work has been found to be of great difficulty and has been directed to:

1. Developing of adequate technic.
2. Preparing for future work.
3. Trying a particular problem—e. g., effect of strychnin and of quinin on hearing.

This work is going on at two universities: (1) At Cornell, under Dr. Culler, where he has the opportunity of consulting Dr. Bentley, professor of psychology, and Dr. Liddell, professor of physiology, both of whom are interested in this subject. (2) At McGill, where Dr. G. Sutherland has carried on corresponding work aided by Professors Tait, Babkin and Collip.

Dr. Charles B. Davenport, of the Carnegie Laboratory of Genetics, reports:

The work during the first year of the Otosclerosis Committee covering the field of the endogenous or genetic factors in otosclerosis has been as follows:

1. Certain families left incompletely studied by Dr. Milles have been further studied.

2. A series of 23 new families living in and about Baltimore, originating in the clinic of Drs. Crowe and Guild, was studied. Combined with Dr. Milles' families this makes 125 families investigated in the field.

3. From the literature the 75 most complete pedigrees have been copied, analyzed and charts prepared for photographing.

4. A tabulation of data by sex and racial stock has been made, also reports of measurements of stature, sitting height, tibia height, arm length, shoulder breadth, pelvic breadth, weight, body build, hand size and proportions, head proportions and chin have been tabulated.

Important Points in Four Theories on Otosclerosis.*

DR. E. P. FOWLER, JR.,
NEW YORK.

DISCUSSION.

DR. J. GORDON WILSON, Chicago: It is of particular interest to have so clear a demonstration of various views of European investigators on otosclerosis, especially Prof. Otto Meyer's latest conception of the genesis of this disease. He apparently believes that an individual focus appears where he claims a spontaneous fracture has arisen. The argument is that an otosclerotic focus is to be regarded as Nature's repair of these existing spontaneous fractures as featured in predisposed individuals.

DR. JACQUES HOLINGER, Chicago: Dr. Fowler says that the essential part in otosclerosis is the ankylosis of the stirrup in the oval window. There are undoubtedly foci in otosclerosis which have no connection with the oval window and never lead to ankylosis. Usually they start in the vicinity of the oval window but ankylosis is a finding in late cases. I think Dr. Fowler will admit that otosclerosis is a late ossification of the cartilage remnants in the capsule of the labyrinth. I do not think this has been disproved yet. Wittmaack is not the only author working on otosclerosis nor the only authority.

DR. EUGENE R. LEWIS, Los Angeles: There are two or three points I would like to make. In regard to the calcium-supplying foods, for example, there is no question that the chemical constitution of many vegetables in different parts of the country differs very materially in electrolytes as well as nonelectrolytes. In at-

*This paper appears in full on page 175 of this issue.

tempting to bring about alteration of calcium in the body we must know something about their local peculiarities of constitution if we are to attempt to bring about such changes by diet.

Second, I think it is unmistakably established that the biochemical constitution of the otosclerotic is different from that of the ordinary individual. He is constitutionally predestined to the disease. These constitutional predispositions seem to follow the Mendelian law of transmission—from father to daughter and from mother to son. Unfortunately, the path of transmission is not always clear; there may be dominants both on the father's and the mother's side. That confuses the picture, making it difficult to determine exactly certain transmissions of hereditary characteristics. Inherited tendencies are not sole factors in producing individual manifestations. Nutrition and pathologic episodes probably play important rôles in the actual development of fixation processes in hereditarily predisposed individuals.

DR. ARTHUR DUEL, New York: We should be very thankful to Dr. Fowler, Sr., for producing this offspring, and to Fowler, Jr., for gathering together these important specimens. I do not think there are any other such specimens available in a collection. This is a highly interesting demonstration.

DR. E. P. FOWLER: There is nothing I can add. My son covered the subject pretty thoroughly. We were able to get these specimens by hook or crook, and some had to be coaxed from their owners. I feel it is an extraordinarily complete collection. Some of the slides are priceless.

DR. WALKER, Boston: I think it a remarkable thing that we can have these slides available in New York.

DR. E. P. FOWLER, JR.: I should like to correct an impression which I seem to have given inadvertently. I am not reporting on all the theories of otosclerosis, merely important points in a few theories.

In regard to the re-ossification of cartilage, it is a perfectly good theory. Incidentally, Wittmaack's resorption takes place in the pseudocartilaginous remnants, and Weber's osteitis fibrosa always begins at the edge of cartilaginous areas.

As for ankylosis of the stapes being identical with otosclerosis, that is the statement of Bruhl, who believes that the condition should be approached only from the clinical viewpoint.

DR. W. P. EAGLETON, Newark, N. J.: What is the strain he spoke of?

DR. E. P. FOWLER, JR.: The tensor tympani pulls around the cochlear process and produces a strain on the "otosclerotic corner." The stapedius and the other ossicles pull upon the stapes, which has a much wider excursion anteriorly than posteriorly. The pull of the tensor tympani and the irritation of the movement of the anterior border of the stapes, according to Bruhl, cause otosclerosis in hereditarily predisposed individuals.

DR. J. GORDON WILSON, Chicago: A very small lesion in a child may increase with development. In course of time the internal auditory meatus in some cases is almost tilted downwards. That is how the mechanism for fracture occurs.

DR. JACQUES HOLINGER, Chicago: There is a recent article by O. Meyer which gives an explanation of the spontaneous microscopic fractures. The temporal pyramid is likened to a console. The base is solid in the wall of the skull; the medial part, which is the point of the pyramid, is more or less loose and movable on account of the furrows in the base of the skull anterior and posterior to the pyramid. Therefore the vibrations of walking, jumping and pounding of the feet on the ground have the effect of working on the tip of the pyramid of the temporal bone and producing those fissures which are always perpendicular to the edge of the pyramid. The fissures are the result of the forces that the base of the skull is regularly exposed to.

Effects of Some Drugs on the Vestibular Responses to Rotation.

DRS. ELLISON L. ROSS, MURLAND W. FISH AND AXEL OLSEN,
NORTHWESTERN MEDICAL SCHOOL,
CHICAGO.

Pilocarpin, nicotin, morphin, camphor, atropin, cocain, nitrite, bromid picrotoxin, apomorphin, caffen and strychnin, in moderate doses, were studied. The work was done on dogs. Duration

of nystagmus after a standardized method of rotation was taken as the test for changes in vestibular response.

No drug stimulated vestibular reaction. Pilocarpin, nicotin, morphin, camphor, atropin, cocain and nitrite gave results so nearly normal that they were considered inactive. Bromid, picrotoxin, apomorphin, caffen and strychnin decreased the duration of nystagmus over 10 per cent (calculated drug action). Strychnin lowered it 21 per cent. The importance of actions of bromid, picrotoxin, apomorphin and caffen is uncertain with the data at hand.

The action of strychnin was considered suggestive. Therefore a depression of the spinal cord was produced by spinal anesthesia with procain in a series of ten dogs. The average of these tests was 32 per cent higher than the average on a corresponding number of tests on normal dogs.

It was considered that the spinal cord had exerted an inhibitory influence on the duration of nystagmus resulting from rotation.

The columns of Goll and Burdach on one side of the cord were cut between the skull and atlas in a series of dogs. Standard rotation tests were made several days following operation. In the large majority of observations the labyrinth opposite the side of the lesion gave the greater response.

DISCUSSION.

PROF. R. L. WEGEL, New York: I noticed in some of the recovery cases that there is a typical dip which is characteristic of all types of nervous reaction. I would like to know whether Professor Ross has correlated these types with the physiology of nerve metabolism.

DR. JOHN GUTTMAN, New York: I would like to know whether Prof. Ross noticed nystagmus during etherization of the animals. Does he explain this by central or peripheral reaction? Also did he notice any deviation in younger or older animals?

DR. WELLS P. EAGLETON, Newark: I would like to know why Prof. Ross made his tests on dogs in an upright position. A dog naturally walks on all fours and never normally assumes the upright position.

PROF. ROSS (closing): I have made no comparison of my curves with those of nerve fatigue curves, so that I cannot speak on that part of the subject at all. As to the ages of the animals used in my experiments, they were not recorded. I have no explanation for nystagmus occurring under anesthesia. However, in this connection it may be of interest for you to know that the majority of my animals showed a spontaneous nystagmus during spinal anesthesia. As to why I chose to rotate animals in the position described, all I have to say is that this was the only method that I was able to find which produced a nystagmus lasting over a long enough period to be easily measurable and to have a minimum percentage of error.

SYMPOSIUM: INTRACRANIAL LESIONS OF OTITIC ORIGIN.

A. From the Otologist's Standpoint.

DR. HARRY P. CAHILL,

BOSTON.

Cahill points out that over 80 per cent of the intracranial complications of middle ear disease result from neglected chronic suppuration. He maintains that even after clearing up the chronic suppuration, the patient with a postmarginal defect or Shrapnell perforation should know how little the otologist can promise him regarding the future. The value of roentgen ray findings in chronic suppuration is in showing the presence of serious pathology rather than in negative findings.

In abscess of the temporosphenoidal lobe the avenue of approach must be either through a bone flap, a temporoparietal decompression, a subtemporal decompression, or through the mastoid wound extended. The latter has given the higher average of recoveries. Lipiodol injection is of value in some cases of brain abscess. It shows how collapse of the abscess wall is taking place and the position of the drain with relation to the abscess capsule.

Osteomyelitis of the petrous apex has usually a latent period for determining whether further surgical intervention is called for. Presence of the symptom complex of localized meningitis,

including septic temperature, beginning stiff neck, slight Kernig's sign with increased cells in the spinal fluid indicate extension from the carious apex into the neighboring cisterna.

B. From the Ophthalmologist's Standpoint.

DR. CONRAD BERENS,

NEW YORK.

Dr. Berens gives the following conclusions:

The brain abscess is frequently ipsilateral with papilledema. The presence of this ocular condition is an aid in the diagnosis in the early stages of the development of the lesion, especially if the intraocular tension is equal in both eyes or greater in the eye showing more pronounced papilledema.

Hemianopsia is of slight value in the differential diagnosis of brain abscess but may be an important aid in localizing the lesion, especially when the condition follows bilateral mastoidectomy. Hemianopsia is also of value as an aid in the diagnosis of temporo-sphenoidal lobe abscess.

Paralysis of the sixth nerve frequently accompanies the complications of diseases of the middle ear and occasionally of the external ear. Permanent paralysis is rare and restoration of aural and ocular functions may be complete without surgical intervention.

SUMMARY.

1. A lesion of the right cerebral hemisphere may be suspected if the quick component of nystagmus to the left is absent or if there is conjugate deviation of the eyes to the right upon labyrinthine stimulation.

2. Theoretically, a lesion destroying the nerve endings in the horizontal canal fibers in the eighth nerve or in the medulla, before the fibers entered Deiter's nucleus, would indicate absence of nystagmus, vertigo and past pointing from horizontal canals on the affected side.

3. Theoretically, a lesion destroying the endings in the vertical canals and the eighth nerve or in the vertical canal tracts in the

pons, before they decussated, sending fibers for vertigo to the cerebrum, denotes absence of vertigo, nystagmus, past pointing and falling from the vertical canals.

4. A lesion may be suspected in the posterior portion of the pons near the posterior longitudinal fasciculus if there is no nystagmus from the vertical canals and stimulation of the vertical canals produces normal past pointing, falling and vertigo.

5. There may be a lesion of the eighth nerve or complete destruction of the labyrinth, if complete deafness occurs unaccompanied by nystagmus, vertigo, past pointing or falling.

6. If there is no nystagmus after stimulation of the horizontal semicircular canals, but vertigo, past pointing and falling, there probably is a lesion of the vestibulo-ocular tract for nystagmus in its passage through the medulla after the fibers for vertigo decussated in Deiter's nucleus and before they enter the posterior longitudinal fasciculus. An irritative lesion in this position might produce perverted nystagmus, as reported by Fisher in the patient in whom a tumor of the left cerebellopontine angle was found at autopsy.

7. If nystagmus is normal but vertigo and past pointing are impaired upon stimulation of the horizontal canals, a lesion of the vertigo fibers after they leave Deiter's nucleus and before they enter the cerebellar nuclei is suspected: Such a lesion would usually involve the inferior cerebellar peduncle and theoretically might be associated with an increase in the duration (Neuman) and amplitude of nystagmus by interfering with the descending impulses of the eye muscle nuclei which are supposed to be carried by the inferior cerebellar peduncle.

8. A lesion of the posterior longitudinal fasciculus is suggested by absence of nystagmus after stimulation of both vertical and horizontal semicircular canals in the presence of normal past pointing, falling and vertigo.

9. Normal nystagmus, but absence of vertigo, past pointing and falling after stimulation of the vertical canals is suggestive of a lesion of the tracts for vertigo, after decussation has occurred in the pons; the lesion may be in the pons, the middle cerebellar peduncle or cerebellum.

10. If stimulation of both the vertical and horizontal semicircular canals produces normal nystagmus, but not past pointing, falling or vertigo, the lesion is probably in the cerebellum, superior cerebellar peduncle, or upper part of the pons.

C. From the Neurologist's Standpoint.

DR. RAMSEY HUNT,

NEW YORK

In my earlier years I was called quite often to the infirmary on consultations, but as the years have gone on there has been less of this work because, I believe, of the greater facility otologists have acquired in the diagnosis of this group of cases. They have made the field of brain abscess their own, and this is right, for it is a chapter in neurological surgery which really belongs to rhinologists and otologists for the reason that these more serious complications arise in the course of the daily routine of these specialties. Not only is diagnosis very difficult in this group of cases, but the decision as to operation is often an even more delicate question, and it requires the finest judgment and a large experience to pass on otitic complications, in which mastoiditis, brain abscess, sinus thrombosis, and meningitis often occur in combination. It is not necessary for me to enlarge on the question of diagnosis to the members of this Association; when the case is clear cut diagnosis is not difficult, but in the atypical or complex types even the most skillful often fail. In temporal lobe abscess, localized on the right side in right handed individuals, when there would be no aphasia, I have often found an important sign, the absence of the abdominal reflex on the affected side, which may be accompanied or followed by hemiparesis, hemisensory disturbances and the Babinski reflex. To this is often added a right optic neuritis, or a greater involvement of the optic disc on the right side. When the abscess is situated on the left side, the early development of aphasia is often quite characteristic.

While in the temporal lobe lesions, the localizing symptoms are usually contralateral, in cerebellar lesions the effects are usually homolateral, giving rise to intention tremor and asyner-

gia, and often involvement of the adjacent cranial nerves, the facial, glossopharyngeal, vagus and hypoglossal.

The general symptoms of brain abscess are often difficult to interpret, because headache, vomiting, rigidity of the neck, may all accompany mastoid disease or sinus thrombosis. Therefore it is not always possible to lay stress on the general symptoms, and more importance is often attached to special pressure symptoms or localizing signs.

I think the greatest advance in the future will be made when the otologist approaches this problem from the neurological standpoint, and it would be of the greatest advantage if you would encourage the younger men, whose destinies you are guiding, to spend a year or two years in a general neurological and neurosurgical clinic.

In your work you have brought your studies to an astounding degree of perfection, especially since the work of Bárány. We do not understand that as well as you do. We do not interpret those tests as well as you do. I believe the time is coming when the whole field of septic meningitis and brain abscess in the anterior, middle and posterior fossae, and their relation to the sinuses and mastoid region, will be in the hands of otologists who are grounded in neurological methods. This, in a way, rings the death knell of the old time neurologist in this field, but it welcomes the appearance of the otoneurologist, or the neurotologist to a more important field.

Pathways of Intracranial Complications.

DR. ISIDORE FRIESNER,
NEW YORK.

It must be obvious that in order to establish a rational basis for the exploration of structures beyond the mastoid we must lay as a foundation a thorough understanding of the pathological changes and the pathways along which these changes progress. Realizing the importance of this problem, we have carefully prepared and studied histologically a considerable number of temporal bones illustrating the various pathways of infection. An infection beginning in the middle ear may extend through va-

rious routes and involve a variety of structures. Some of the possible routes are by (1) bone necrosis, (2) through sutures, (3) dehiscences, (4) perforating vessels, or (5) lymphatic. In this presentation, examples are furnished of otitic complications, with brief histories of the cases, and histological sections to demonstrate the pathway of the spread of the infection.

The slides beautifully demonstrate the *modus operandi* in the following cases: (1) left temporal lobe abscess secondary to zygomatic perforation; no mastoiditis; (2) mastoiditis, extradural abscess, temporosphenoidal lobe abscess meningitis; (3) otitis media, with scarlet fever mastoiditis, cerebellar abscess; (4) otitis media, mastoiditis, osteitis of the petrous pyramid, meningitis; (5) osteitis of the petrous pyramid, deep subdural abscess, meningitis; (6) meningitis with retrograde labyrinthitis; (7) otitis media, labyrinthitis, meningitis; (8) otitis media, mastoiditis, sinus thrombosis, meningitis; (9) mastoiditis epidural abscess meningitis in a type three pneumococcus invader.

In conclusion we hope that presentations of this nature will stimulate further studies along similar lines, which to date have not been plentiful.

DISCUSSION

SIR CHARLES BALLANCE, London: I am grateful to you for your kind welcome to me on this second occasion of my visit to you. I am glad to be here today. Before I came to this meeting, I was promised a copy of the papers that have been presented so that I might discuss them intelligently. This promise was not carried out.

However, I have been impressed by the splendid papers we have had today and also the number of cases which have been carefully reported. If I had read these papers beforehand, I might have been able to appreciate the details of the clinical cases and to present to you a fair statement of my views on these subjects, but I do not feel competent to discuss the papers until I have read and thought over them. I wish to say how much I have appreciated the opportunity of listening to the opening papers and how gratefully I shall read them when they are in print.

Therefore, instead of doing as you expected me to do, I will do instead what you did not expect me to do and tell you some incidents of my life.

My first paper on intracranial complications, four cases of lateral sinus pyemia, was presented in 1890. These cases made an immense impression on me. There was one point in that paper that I think is still true—that is, I urged that the surgeon should decide before operation whether or not there was an infection of the lateral sinus. If the sinus was infected, I thought it was of great importance to tie the vein before operating on the temporal bone lest the latter operation should displace infected clots into the lungs. Fifty years ago I emphasized that this was the proper treatment.

If I had to make a statement as to one thing that was most important to us in practice, I should say that the *clinical examinations* are more important than anything else. You may call in a laboratory man, a neurologist, an ophthalmologist or other specialists, but you cannot delegate your responsibility and you can not operate unless you feel personally commissioned by your own examinations to do the operation. Examinations and notes should be made on the patients twice a day. The best method on a difficult case is personal examination of the patient morning and evening. A sign may not be there in the morning, but on evening examination it may be present.

Another point which is important to remember is that a symptom not observed is not necessarily a symptom not present.

There is another method of importance which is not used as much as it should be, that is, the use of the perimeter. In temporal lobe abscess it may be the only thing at the moment that may determine the site of the abscess. If the typical defect is present in the visual fields, immediate operation is justified.

In regard to chronic otorrhea, there are a few cases with slight headache and optic neuritis. In regard to temporosphenoidal abscess, I do not think I can contribute anything new to the discussion. If there is a stabile pupil on the side of the discharging ear, operation is justified.

Another sign of great importance is alteration in the sense of smell. This sign has been of great help in making a diagnosis. A man sent up to my ward at St. Thomas Hospital was a gardener by trade. He told me he could not distinguish the smell of the rose from that of the violet. I operated on that man that very afternoon and found a large quantity of pus in the temporal lobe. Another sign which is diagnostic is the dream state. Hughlings Jackson noted this sign. He had a case where a woman would sit up in bed staring vacantly in a dream state, but when anyone passed, she would exclaim, "What a horrible smell!" This is diagnostic of temporosphenoidal abscess or tumor.

I would like to recall the work of Preysing, who sawed the heads of his fatal cases transversely and longitudinally and showed that temporosphenoidal abscess was attached by a stalk and that drainage could be made through the stalk, which obviates hernia cerebri and meningitis. I believe this is one of the greatest advances in the treatment of brain abscess.

Two other points may be mentioned, one is that malaria simulates all sorts of diseases. I have known a case with discharge from the ear diagnosed as lateral sinus pyemia which was cleared up by intravenous injection of quinine.

Another class of case which is deceiving is the alternating facial palsy which may occur in association with a discharging ear in children with marasmus. This may lead the unwary to diagnose temporosphenoidal abscess.

DR. WELLS P. EAGLETON, Newark, N. J.: Probably this is the greatest compliment that has ever been paid me, that I am associated in this discussion with Sir Charles Ballance. I am very happy to recall to my mind the things he has contributed to my knowledge of this subject and to the progress of otology. One of the first papers he published in the Transactions of St. Thomas Hospital was on the cure of cerebellar abscess.

There is another point I should like to mention and that is about Andrew Pare; in 1530 he described a case of temporosphenoidal abscess. He said something that was forgotten until 1925. He put in a lead pipe and then he had the patient close

his nose and his mouth and blow to increase the intracranial pressure.

I want to record another thing which I have never reported before and this is that every case of hernia cerebri in which the patient got maggots recovered. That means we do not touch the cases for a long time. Last week I proposed to put sterile maggots in to see the result. We had a case of osteomyelitis of the base of the skull in which there were maggots. It did marvelously.

Work on the petrous pyramid and the temporal bone has shown that the temporal bone is a capsular bone and that the inner surface of it is median (?) bone the same as any other bone. It is like the growing end of the long bones. It joins another peculiar bone, and that is the sphenoid base which has a growing marrow. Dr. Kopetsky says that under the petrous apex one may find pneumatic cells. In disease of the petrous apex or the base of the sphenoid we have a true homogeneous osteomyelitis. If you open the sinus and find the infection, the bloodstream infection can be cured. If you can't find a method of getting through the inferior petrosal the chances are it is osteomyelitis of the petrous apex or base of the sphenoid that produces the blood stream infection without sinus thrombosis.

As regards optic neuritis, we have learned there are two different conditions. If you tie the jugular you cut off the return blood from the head. Optic neuritis occurs, but it is slight. The chances are you have interfered with the return flow of blood from the base to the brain itself. But if there is a sinus thrombosis this involves the branches of the vein of Galen into the central part of the ventricular system, and you have a distension of the ventricles, causing papilledema. This is the condition that we find in brain abscesses.

DR. JOHN MCCOY, New York: One point strikes me in this discussion. Fifteen years ago I had a patient, a boy of 12. He had a radical frontal sinus operation. He subsequently developed lower mentality and slow pulse, from which we made a diagnosis of brain abscess. I operated on the boy and the temperature went up to 106. I decided there was insufficient drainage.

I put an opening into the side of the head, after which the temperature came down and the boy got well. Two years ago I had another patient with slow pulse and other signs of brain abscess. I operated on him and decided there was temporosphenoidal abscess. I explored from one end to the other without result. I could not locate a brain abscess. The patient developed meningitis and died some ten days later. At autopsy the abscess was found in the posterior portion of the frontal lobe, and we were getting effects from pressure backwards rather than forwards. Therefore, if you fail to locate the abscess in the temporosphenoidal lobe, the search should be made further forward.

DR. H. P. CAHILL: I do not feel that I wish to add anything to this discussion. You have heard Sir Charles Ballance and Dr. Eagleton give the result of their experience, and I do not wish to disturb the memory of these two discussions.

(To Be Continued in the June Issue.)

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